U.S. Department of Labor

Office of Administrative Law Judges
800 K Street, NW, Suite 400-N
Washington, DC 20001-8002

(202) 693-7300
(202) 693-7365 (FAX)

Issue Date: 14 February 2006

CASE NO.: 2004-BLA-00050
2004-BLA-05354

In the Matter of:

MILDRED E. BURNS, survivor of
and on behalf of THOMAS BURNS,
Claimant

v.

CONSOLIDATION COAL CO.,
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-in-Interest

Appearances:

Sandra M. Fogel, Esq.
For the Claimant

Gary B. Nelson, Esq.
For the Employer

Before: Stephen L. Purcell
Administrative Law Judge

DECISION AND ORDER – AWARDING BENEFITS

This proceeding arises from a miner's subsequent claim filed by Thomas Burns ("Miner"), and a survivor's claim filed by Mildred E. Burns ("Claimant" or "Widow") under Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. § 901 et seq. ("the Act"). Benefits under the Act are awarded to coal miners who are totally disabled due to pneumoconiosis. Surviving dependents of coal miners whose deaths were caused by pneumoconiosis may also recover benefits. Pneumoconiosis, commonly known as black lung, is a chronic dust disease of the lungs arising from coal mine employment. 20 C.F.R. § 718.201 (2004).

The Act's implementing regulations are located in Title 20 of the Code of Federal Regulations, and section numbers cited in this decision exclusively pertain to that title. References to "DX," "CX," and "EX" refer to the exhibits of the Director, Claimant, and Employer, respectively. The transcript of the hearing is cited as "Tr." and by page number.

On November 19, 2003, these cases were referred to the Office of Administrative Law Judges for a formal hearing. (DX 76). Following proper notice to all parties, a hearing was held on April 14, 2005 in Carbondale, Illinois. DX 1-57 and 59-76 were admitted into evidence pursuant to 20 C.F.R. § 725.456. A ruling on the admissibility of DX 58 (also marked by Employer as "EX D") was reserved pending the filing of written briefs by the parties. Tr. 14. CX 1-8 in support of the Miner's claim and CX 5, 7-8 in support of the Widow's claim were similarly admitted into evidence. Tr. 14-15. EX 15-18 and 21-22 were admitted in the Miner's claim and EX A-C and F-I were admitted in the Widow's claim, while a ruling on the admissibility of EX 11-14, 19-20, and D-E was reserved pending the submission of written briefs by the parties.

In my post-hearing order issued July 25, 2005, I excluded DX 58/EX D from the Widow's claim but admitted that exhibit in the Miner's claim. I also excluded EX 14, 21, E, and H in the Widow's claim but admitted those exhibits in the Miner's claim. Finally, EX 13 and 22 were admitted in the Miner's claim. I further ordered that evidence admitted into the record in the Miner's claim would not be considered in deciding the Widow's claim.

The parties thereafter had full opportunity to present closing arguments in the form of post-hearing briefs. Claimant's and Employer's closing arguments were both received on September 22, 2005. However, after reviewing the parties' briefs, and the evidentiary record, I concluded that several evidentiary issues remained unresolved, and I conducted a telephone conference with counsel for the parties on November 7, 2005 to resolve those issues.

During the telephone hearing, I first noted that the February 12, 2003 medical report of Dr. Crouch was not among the exhibits produced by Employer either before or during the formal hearing, although the report was referenced by both parties in their post-hearing briefs. I thus directed Employer's counsel to forward a copy of the report to me for inclusion in the record as EX 21B. I further noted that Dr. Tuteur's report, marked as EX 21, would be marked as EX 21A, and that both exhibits were admitted in evidence.

Second, I noted that, contrary to my earlier indication that DX 58 was not included among the Director's exhibits forwarded to the Office of Administrative Law Judges by the District Director, that exhibit was contained in Claimant's file and consisted of several hundred pages of documents which included EX 4 through 10.

Third, I admitted in the Miner's claim EX 11, the curriculum vitae and chest x-ray interpretations by Dr. Wiot of November 4, 1986 and November 22, 1999 chest x-rays, as well as EX 12, Dr. Naeye's December 4, 2003 medical report supplementing his earlier April 20, 2003 report. No specific ruling on the admissibility of these two exhibits had previously been rendered in light of Claimant's counsel's incorrect representation that they were included among the documents contained in DX 58.

Fourth, I directed the parties to prepare and submit to me within fourteen days written statements outlining their positions regarding Employer's request that it be permitted to substitute in the Widow's claim Dr. Naeye's medical report, identified both as EX D and DX 58 (EX 4), for the May 6, 2004 medical report of Dr. Fino identified as EX I.

Fifth, I advised the parties' attorneys that the Pulmonary Function Tables found at 29 C.F.R. Part 718, Appendix B, did not contain pulmonary function values for individuals over 71 years of age and that I therefore intended to obtain from the Office of Workers' Compensation Programs, and forward to both parties, information regarding computing the values for the Miner who died at age 79.

On November 21, 2005, I received a Joint Brief in Support of Election of Evidence signed by counsel for both parties. In that pleading, the parties agreed that Dr. Naeye's reports dated April 20, 2003 and December 4, 2003 (EX D and EX J) could be substituted for Dr. Fino's report (EX I) in the Widow's claim. The pleading further notes that neither party addressed Dr. Naeye's reports in their post-hearing briefs, and they therefore requested that their previously filed briefs be stricken from the record and that a new briefing schedule be established.

On November 29, 2005, in response to the parties' joint stipulation and request, I issued an order allowing the substitution of EX D and EX J for EX I as evidence in the Widow's claim, directing counsel to file simultaneous written closing briefs within forty-five (45) days, and striking their previously filed written arguments. Briefs were subsequently filed by both parties and have been considered in reaching the decision herein.

The Findings of Fact and Conclusions of Law that follow are based upon my analysis of the entire record, the arguments of the parties, and the applicable regulations, statutes, and case law. They also are based upon my observation of the demeanor of the witnesses who testified at the hearing. Although perhaps not specifically mentioned in this decision, each exhibit and argument of the parties has been carefully reviewed and thoughtfully considered. While the contents of certain medical evidence may appear inconsistent with the conclusions reached herein, the appraisal of such evidence has been conducted in conformance with the quality standards of the regulations.

ISSUES

The only issue that remains for resolution in the Miner's claim is whether the miner was totally disabled due to pneumoconiosis. The only issue remaining in the Widow's claim is whether the Miner's death was due to pneumoconiosis. DX 76; Tr. 5-6.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Factual Background and Procedural History

The Miner first filed for benefits on September 3, 1986. DX 32 at 45-48. The claim was informally denied on January 23, 1987 because the Miner had failed to establish any element of

entitlement. *Id*. at 4-5. No appeal of that decision was filed and the denial thereafter became final. *Id*. at 1.

The Miner filed a subsequent claim for benefits on October 22, 1999. DX 1. The claim was informally denied on March 3, 2000 because the Miner had failed to establish that he was totally disabled. DX 11. The Miner thereafter requested a hearing, DX 13, and the case was referred to the Office of Administrative Law Judges on January 3, 2001. DX 34. The Miner died on June 7, 2002 while his claim was pending before this office. DX 38.

On July 15, 2002, the Widow filed a claim for survivor's benefits. DX 36. On February 27, 2003, Administrative Law Judge Robert L. Hillyard, at Claimant's request, remanded the Miner's claim to the District Director so that it could be consolidated with the Survivor's claim and evidence could be developed in both cases. DX 53. On September 23, 2003, the District Director issued a proposed decision and order awarding benefits. DX 69. Employer thereafter filed a timely request for a hearing. DX 71. The consolidated claims were then forwarded to the Office of Administrative Law Judges for hearing on November 19, 2003. DX 76.

As noted above, a formal hearing was held before me on April 14, 2005 in Carbondale, Illinois. Claimant and her daughter, Bonnie Lee Dawe, testified at the hearing.

Ms. Dawe testified that she is the postmaster in Elkville, Illinois, is 58 years of age, and is the daughter of Claimant and the deceased Miner. Tr. 27. She lived with her parents until age 19 and routinely observed her father covered with coal dust when he would come home from working in the mine. Tr. 28. By the late 1970's she noticed that he was no longer able to play ball with her brothers. *Ibid*. He became frail, lost weight, had problems breathing, and limited his activities around the house. Tr. 28-29. When he retired from mining in 1985, his health was not good and he could only walk about four blocks before having to rest. Tr. 29. His breathing became more labored and he was more frail by the 1990's. Tr. 30. He began using oxygen at home after he was hospitalized for heart surgery. *Ibid*. He was 74 when he had his heart surgery in 1997. Tr. 31. He was reluctant to use oxygen because he was very proud and did not want to be sick. *Ibid*. He had a lung biopsy in 1999 which was performed by Dr. Crabtree prior to a hernia operation. Tr. 31-32. He also had a chest x-ray performed after he broke his hip on March 1, 2002 and was told by the doctor that he had lung cancer. Tr. 32. He stopped smoking cigarettes around mid 1953. Tr. 34. He had a valve replacement and quadruple bypass surgery in 1997. Tr. 34. He did not wake up for three days after the surgery, and it was then that they found out he had lung problems. Tr. 35. He was involved in a serious automobile accident in 1989 and suffered a broken rib which "went into his lungs." *Ibid*.

Claimant testified that she and the Miner were married at the time he passed away. Tr. 39. They knew each other when they were children and attended the same schools. *Ibid*. He joined the Navy after Pearl Harbor was bombed. Tr. 40. He did not smoke before going into the service but was smoking when he returned from the Navy in 1945. *Ibid*. She smoked also but they both quit in 1953. Tr. 41. He had breathing problems when the children were young. *Ibid*. By the 1990's, he was having trouble doing yard work. Tr. 42. The last few years before he died, he would play pool at the recreation center and then rest in the afternoon. *Ibid*. The last year of his life he spent most of his time sitting in a chair at home. Tr. 43.

Coal Mine Employment

The duration of a claimant's coal mine employment is relevant to the applicability of various statutory and regulatory presumptions. Claimant bears the burden of proof in establishing the length of his coal mine work. *See Shelesky v. Director, OWCP*, 7 BLR 1-34, 1-36 (1984); *Rennie v. U.S. Steel Corp.*, 1 BLR 1-859, 1-862 (1978). On his application for benefits, Mr. Burns alleged 40 years of coal mine employment. DX 1. The Employer conceded 40 years of coal mine employment, 13 years of which was underground. After a review of the record, I accept the Employer's concession and find that Mr. Burns has established 40 years of coal mine employment.

Responsible Operator

The Employer has not contested that it is the responsible operator in this case. An employment history form completed by the Miner shows that Mr. Burns worked for Consolidation Coal Company from 1962 to 1985. DX 2. The Social Security records confirm that the Miner worked for Union Electric Development Corporation from 1945 to 1958, Trax Fraer Coal Company from 1958 to 1962, Consolidation Coal Company from 1962 through 1985, and any employment that followed was not coal mine employment. DX 37. Consequently, I find that Consolidation Coal Company is the properly designated responsible operator.

DISCUSSION AND APPLICABLE LAW

A subsequent claim for benefits "shall be denied unless the claimant demonstrates that one of the applicable conditions of entitlement . . . has changed since the date upon which the order denying the prior claim became final." 20 C.F.R. § 725.309(d). To be entitled to benefits under Part 718, a claimant must establish by a preponderance of evidence that (1) he has pneumoconiosis, (2) the pneumoconiosis arose from his coal mine employment, (3) he is totally disabled, and (4) the total disability is due at least in part to pneumoconiosis. *Gee v. M.G. Moore & Sons*, 9 BLR 1-4 (1986).

Medical Evidence

A. X-Ray reports[1]

Exhibit	Date of X-ray	Date of Reading	Physician/ Qualifications	Interpretation
DX 32	11/4/86	11/26/86	Cole, B, BCR	1/0
DX 32	11/4/86	11/6/86	Hummel, BCR	Negative
DX 34C	11/4/86	9/26/01	Westerfield, B	1/1
EX 11	11/4/86	12/10/03	Wiot, B, BCR	Negative
DX 28	11/4/86	10/20/00	Forry, B, BCR	Negative

[1] A chest x-ray may indicate the presence or absence of pneumoconiosis. 20 C.F.R. § 718.102(a), (b). It is not utilized to determine whether the miner is totally disabled, unless complicated pneumoconiosis is indicated wherein the miner may be presumed to be totally disabled due to the disease.

CX 1	11/9/99	12/9/03	Cohen, B	Unreadable
EX 19	11/9/99	4/29/04	Wiot, B, BCR	Unreadable
DX 8	11/22/99	1/3/00	Gaziano, B	1/0
DX 34A	11/22/99	7/13/01	Ahmed, B, BCR	2/1, Category A
DX 34A	11/22/99	7/11/01	Cappiello, B, BCR	3/2, Category A
DX 34C	11/22/99	9/26/01	Westerfield, B	1/1, old trauma, CABG
DX 28	11/22/99	10/20/00	Forry, B, BCR	1/1
EX 11	11/22/99	12/10/03	Wiot, B, BCR	Negative
CX 2	4/6/00	12/9/03	Cohen, B	Unreadable
EX 19	4/6/00	4/29/04	Wiot, B, BCR	Unreadable
CX 3	9/12/00	12/9/03	Cohen, B	1/1
EX 19	9/12/00	4/29/04	Wiot, B, BCR	Unreadable
CX 4	10/13/00	12/9/03	Cohen, B	1/1
DX 34B	10/13/00	8/21/01	Miller, B, BCR	1/2
DX 34B	10/13/00	8/24/01	Ahmed, B, BCR	2/2, Category A
DX 34B	10/13/00	8/20/01	Cappiello, B, BCR	2/2
DX 26	10/13/00	10/13/00	Glazier	No finding of CWP
EX 19	10/13/00	4/29/04	Wiot, B, BCR	Negative

B. Pulmonary Function Studies[2]

Exhibit/ Date	**Physician**	**Age/ Height[3]**	**FEV_1**	**FVC**	**MVV**	**FEV_1/ FVC**	**Comments**
DX 32	Thomson	63	3.17	3.97	100.8	80%	Miner's cooperation and ability to understand and follow instructions good.
11/4/86		63					
DX 58	Schneider	74	2.58	4.11	74	63%	Normal FVC; moderately decreased FEV1; moderately decreased FEV1 percentage; mild improvement in FEV1 of 9% after bronchodilators; lung volumes reveal normal total lung capacity, normal residual volume, normal residual volume
7/16/97		75	2.80	4.20	91	67%	

[2] The pulmonary function study, also referred to as a ventilatory study or spirometry, indicates the presence or absence of a respiratory or pulmonary impairment. 20 C.F.R. § 718.104(c). The regulations require that this study be conducted three times to assess whether the miner exerted optimal effort among trials, but the Board has held that a ventilatory study which is accompanied by only two tracings is in "substantial compliance" with the quality standards at § 718.204(c)(1). *Defore v. Alabama By-Products Corp.*, 12 BLR 1-27 (1988). The values from the FEV_1 as well as the MVV or FVC must be in the record, and the highest values from the trials are used to determine the level of the miner's disability.

[3] The Miner's height was reported variously as 63 to 75 inches. The single study noting the Miner's height as 63 inches is obviously in error and will be ignored. For purposes of determining qualifying disability values, I find that the Miner's height equals 74 inches based on the fact that the vast majority of the studies reflect the Miner's height as 74 inches.

Exhibit/ Date	Physician	Age/ Height[3]	FEV_1	FVC	MVV	FEV_1/ FVC	Comments
							percentage and moderate to severe decrease in diffusing capacity with mild increase in airway resistance. Mild to moderate obstructive airway disease compatible with emphysema.
DX 34A	Crabtree	76	1.27	1.92	34	66%	Severe reduction in FEV1 and FVC with minimal post bronchodilator response; lung volumes show increase in residual volume with low normal total lung capacity show very mild hyperinflation; diffusing capacity markedly reduced. Study suggests combined obstructive and restrictive defect with severe obstruction and moderate restriction with severe reduction in diffusing capacity consistent with diagnosis of CWP.
10/6/99		75	1.35	2.02	27	67%	
DX 5	Sanjabi	77	2.28	3.96		58%	
11/22/99		74	2.48	3.98		62%	
DX 19	Crabtree	77	1.92	3.18		60%	
11/9/99		75	1.98	3.50		57%	
DX 19	Crabtree	77	1.91	3.55		54%	
4/6/00		73	1.90	3.49		54%	
DX 19	Crabtree	77	2.15	3.46		62%	
4/27/00		73					
DX 58	Crabtree	77	1.73	2.91		60%	
9/12/00		74	1.84	3.08		60%	
DX 26	Tuteur	77	1.60	2.71	60	59%	
10/13/00		71.5	1.81	2.62		69%	
DX 58	Crabtree	77	2.33	3.33		70%	
10/26/00		74	2.09	2.79		75%	
DX 34A	Crabtree	77	1.84	2.51		73%	
1/11/01		74					
DX 34A	Crabtree	78	1.95	3.06		64%	

Exhibit/ Date	Physician	Age/ Height[3]	FEV_1	FVC	MVV	FEV_1/ FVC	Comments
2/22/01		74					
DX 34A	Crabtree	78	1.92	2.79		69%	
4/6/01		74					
DX 34A	Crabtree	78	1.16	2.94		40%	
6/21/01		74					
DX 58	Crabtree	78	1.98	2.83		70%	
7/5/01		74					
DX 58	Crabtree	78	1.65	2.56		64%	
8/2/01		74					

C. Arterial Blood Gas Studies[4]

Exhibit	Date	Physician	pCO_2	pO_2	Resting/ Exercise	Comments
DX 32	11/4/86	Thomson	40.6	90.0	Resting	Exercise contraindicated.
			--	--	Exercise	
DX 34A	10/6/99	Crabtree/Moses	44.1	46	Resting	
			--	--	Exercise	
DX 58 (EX 6)	10/22/99	Moses	44.1	46	Resting	
			--	--	Exercise	
DX 7	11/23/99	Sanjabi	39.1	77.0	Resting	Exercise ergometer (complex pulmonary stress) 7 min., 10 sec.
			37.4	84.7	Exercise	
DX 26	10/13/00	Tuteur	38.0	78.0	Resting	Dyspnea on walking less than 100 yards; productive cough; rode bike for 5 min. up to 50 watts.
			34.0	68.0	Exercise	
DX 58 (EX 5)	3/1/02	Coello	44.7	72.0	Resting	
			--	--	Exercise	
DX 58 (EX 5)	4/18/02	Salmon/Migone	40.0	115.0	Resting	
			--	--	Exercise	
DX 58 (EX 5)	6/7/02	Migone	36.4	46.2	Resting	
			--	--		
DX 58	6/7/02	Coello	32.1	48	Resting	

[4] Blood gas studies are performed to detect an impairment in the process of alveolar gas exchange. This defect will manifest itself primarily as a fall in arterial oxygen tension either at rest or during exercise. 20 C.F.R. § 718.105(a).

Exhibit	Date	Physician	pCO_2	pO_2	Resting/ Exercise	Comments
(EX 5)			37.8	51	Exercise	

D. CT Scan Evidence

Dr. Gleason interpreted an October 6, 1999 CT scan as showing extensive abnormal lung parenchymal consolidation involving both lower lobes with associated small to moderate left pleural effusion and tiny right effusion. DX 34A. He noted an approximately 3.5 X 2 cm nodular opacity abutting the paravertebra in the posteromedial right lung base, noting that the possibility it represented a malignant neoplasm could not be excluded. He also noted a linear opacity in the left upper lobe unchanged in comparison to chest films dated back to August 1997 which most likely represented chronic atelectasis and/or scarring.

Dr. Wiot interpreted the October 6, 1999 CT scan as showing no evidence of coal workers' pneumoconiosis. EX 20. He noted the lung fields were markedly over-expanded consistent with emphysema and that the Miner had previous coronary by-pass surgery and apparent insertion of a prosthetic aortic valve. He further noted the Miner showed marked pulmonary edema with a large left pleural effusion and smaller right pleural effusion.

Dr. Meeks interpreted a November 24, 1999 CT scan as showing bilateral emphysematous scarring, emphysematous blebs of the lungs, and left pleural thickening. DX 34A.

Dr. Wiot interpreted the November 24, 1999 CT scan as showing pulmonary congestion, edema and right pleural effusion had resolved. EX 20. He noted that the left pleural effusion had improved and there was atelectatic change in the anterior segment of the left upper lobe which was not a manifestation of coal dust exposure. He also noted similar disc atelectasis on the right and no evidence of coal workers' pneumoconiosis.

Dr. Bean interpreted an April 27, 2000 CT scan as showing diffuse emphysematous changes in both lungs and complete clearing of the previously noted parenchymal process in the posterior lungs bilaterally. DX 19. He further noted scarring within the lungs which was unchanged from the previous exam, resolution of the previously seen pleural effusions, and a small infra renal aortic aneurysm.

Dr. Wiot interpreted the April 27, 2000 CT scan as showing similar findings to the CT scans dated October 6, 1999 and November 24, 1999. DX 20. He also noted that the lung fields were markedly over-expanded consistent with emphysema and showed no evidence of coal workers' pneumoconiosis.

Dr. Menias interpreted an October 13, 2000 CT scan as showing marked diffuse emphysematous changes throughout the right and left lung, most prominent in the upper lobes. DX 26. He noted diffuse scarring along the minor fissure in the ingula and basilar segments of the left lower lobe associated with pleural thickening and two small ill-defined pulmonary

nodules identified in the right upper lobe and right middle lobe which remained indeterminate. He also noted no significant interstitial lung disease or evidence of pulmonary fibrosis.

Dr. Wiot interpreted the October 13, 2000 CT scan as showing no evidence of coal workers' pneumoconiosis. He noted the lung fields were over-expanded consistent with emphysema and a linear scar in the left upper lung field with associated pleural disease consistent with a past inflammatory process but not a manifestation of coal dust exposure. He further noted a linear scar on the right with associated small bullae adjacent to it and severe arteriosclerotic change in the thoracic area.

Dr. Cohen interpreted the October 13, 2000 CT scan as showing diffuse irregular and round opacities between 1.5 mm and 3 mm in diameter scattered throughout the lungs and diffuse emphysema. CX 5. He also noted there were no nodules greater than 1 cm.

Dr. Wiot interpreted a January 10, 2002 CT scan as showing no evidence of coal workers' pneumoconiosis. EX 20. He noted that the lung fields were markedly over-expanded consistent with emphysema and that there were several areas of atelectasis. He further noted there was a prominent right pleural effusion, less significant left pleural effusion, and evidence of pulmonary congestion with an enlarged heart.

E. Biopsy/Autopsy Evidence

On October 6, 1999, Dr. David Crabtree performed a fiberoptic bronchoscopy of the Miner at St. Johns Hospital in Springfield, Illinois. DX 58 (EX 6). The operative report notes that the trachea and endobronchial tree were examined, lavaged, brushed and then biopsied with forceps. The surgical pathology report reflects a microscopic pathologic diagnosis of endobronchial tissue with edema, mild chronic inflammation, and reactive epithelial changes. The specimen did not contain alveolar tissue, and there was no evidence of neoplasm. The cytology reports from that procedure noted that no malignant cells had been identified.

An autopsy of the Miner was performed on June 7, 2002 by Dr. Patrick O'Neill, limited by the Miner's wife to the lungs. DX 41. Dr. O'Neill noted that the deceased Miner appeared his stated age of 79 and, with respect to his internal exam, wrote:

> Most significant is the extensive fibrinous adhesions of the pulmonary pleural surfaces bilaterally. After removal, the lungs weigh 550 grams (right) and 223 grams (left). It is difficult to delineate the anatomy of the left lung due to the extensive fibrinous adhesions. The right lung shows a consolidated area in the lower lobe. A culture of this area is performed. The pulmonary arteries bilaterally are enlarged, but are free of thromboemboli. There is marked black anthracotic pigment deposition in both lungs. There is no significant pleural effusion. Metallic clips are identified on the midline chest plate, again, consistent with history of aortic valve replacement.

Ibid. With respect to his microscopic examination, Dr. O'Neill wrote that the left lower lobe of the lung showed fibrosed granulomas with central necrosis and large numbers of histiocytes

containing black anthracotic pigment within the fibrous tissues. There was also dilation of alveolar spaces in the right and left lung tissues representing emphysematous change and scattered fibrous nodules containing intense anthracotic pigment. Dr. O'Neill noted slender white crystals on polarization consistent with silica and stated that the consolidated areas of the right lower lobe showed fibrin, neutrophils and lymphocytes filling, expanding and replacing the alveolar spaces representing a pneumonic process. His final diagnoses were coal workers' pneumoconiosis, pneumonic consolidation in the right lower lobe, marked pleural adhesions bilaterally, dilated pulmonary arteries bilaterally, findings consistent with clinical history of severe chronic obstructive pulmonary disease ("COPD"), and a 17.0 cm midline chest scar consistent with clinical history of aortic valve replacement.

F. Narrative Medical Evidence

Dr. Douglas Thomson

Dr. Thomson examined the Miner on behalf of the Department of Labor on November 5, 1986, obtained family, medical and employment histories, and conducted objective tests including a chest x-ray, pulmonary function study, and arterial blood gas test. DX 32. He noted a smoking history from 1941 to 1958 of 1 pack per day, and medical history of arthritis, heart disease, diabetes, and high blood pressure, with complaints of dyspnea on climbing and angina when he gets excited. Dr. Thomson diagnosed mild silicosis not related to dust exposure based on chest x-ray with no functional significance.

Dr. Parviz B. Sanjabi

Dr. Sanjabi performed an examination of the Miner on behalf of the Department of Labor on November 23, 1999. DX 6. He obtained family, medical and employment histories, and conducted objective tests including a chest x-ray, pulmonary function study, and arterial blood gas test. He noted a history of pneumonia, pleurisy, wheezing, arthritis, heart disease, diabetes and high blood pressure, and a smoking history of one pack per day beginning at age 21 and ending in the 1950's. Physical examination revealed sputum production, dyspnea, cough, and hemoptysis. Dr. Sanjabi diagnosed coal workers' pneumoconiosis, history of coronary artery disease, a "scar on lung" possibly due to past trauma, and chronic bronchitis by history. He attributed the coal workers' pneumoconiosis to the Miner's prior exposure to coal dust and rated his respiratory impairment as "severe."

On April 13, 2000, Dr. Sanjabi authored a supplemental report in which he responded to a request for additional information by David Marchand, a Senior Claims Examiner for the Department of Labor, as follows:

> As you have indicated in your notes, the pulmonary function test in this man is unusually abnormal. We had no other cause to attribute this abnormality other than to the coal worker's pneumoconiosis, in which he had a good long history of exposure.

> The question posed is whether or not this man is totally disabled. Although his pulmonary function test as you have indicated does not meet the actual standard of disability, I believe the work performed on metabolic testing should be considered as a more dependable measurement. When we had to discontinue the test on this gentleman, the total METS performed was not over 4. This is far below the average required METS for the coal worker's pneumoconiosis [sic] in the fields, which is usually in the range of 6.9 to 7.2. Thus I have to believe that although he has somewhat better pulmonary function test results than a disability would require, his actual physical ability to perform is altered based on the data I have provided for you. . . . .

DX 17.

Dr. David C. Crabtree

Dr. Crabtree wrote, in a one-page letter dated April 27, 2000, that Thomas Burns was his patient and was being evaluated for long-standing shortness of breath and abnormal chest x-rays with recurrent hemoptysis. DX 19. Dr. Crabtree wrote that the Miner had been found to have significant bronchiectasis and bronchial obstructive lung disease, and a significant defect noted on pulmonary function tests since October 1999. He also had chronic dyspnea and was "unable to do much activity whatsoever because of his significant abnormality." *Ibid*. Dr. Crabtree opined that the Miner's condition was "all consistent with his previous exposure in the workplace in the coal mine, consistent with coal worker's black lung." *Ibid*.

In another one-page letter dated October 29, 2002, Dr. Crabtree wrote that the Miner, who had then recently passed away, had a known history of severe, end stage, pneumoconiosis, significant bronchiectasis, and chronic pulmonary fibrosis. DX 42. He further wrote:

> He, without a doubt, had significant underlying lung disease; and while I did not take care of him during the last illness prior to his death, his death in all likelihood is directly related to his chronic underlying lung disease which was baseline end stage when I first met him and had progressed slowly over the ensuing years.

Ibid. He further noted that the Miner's death was related to his chronic coal workers' pneumoconiosis. *Ibid*.

Dr. Peter Tuteur

The Miner was examined at Employer's request by Dr. Tuteur on October 13, 2000. DX 26. Dr. Tuteur obtained relevant work, social and medical histories, and performed a chest x-ray, CT scan, pulmonary function test and arterial blood gas study. Based on all the evidence, Dr. Tuteur concluded that the Miner had extensive intrathoracic disease but no significant coal workers' pneumoconiosis or any other coal mine dust induced disease process. With respect to the Miner's intrathoracic disease, he wrote:

> This is predominantly post inflammatory pleural and parenchymal disease in association with motor vehicle accident, recurrent pneumonias, and coronary artery bypass grafts, associated with aortic valve replacement. Almost certainly he has an endobronchial inflammatory process manifested in part by the history of recurrent bronchitis.

DX 26 at 4. He also noted that the Miner may have pathologically identifiable coal workers' pneumoconiosis, but further noted that, "[i]f so, it would be of insufficient severity and perfusion to produce clinical symptoms, physical abnormalities, physiologic impairment, or radiographic changes including high-resolution CT scan." *Ibid*. While he recognized that the Miner was totally and permanently disabled, Dr. Tuteur concluded that his disability was unrelated to, aggravated by, or caused by his inhalation of coal mine dust or the development of coal workers' pneumoconiosis. *Ibid*.

Dr. Tuteur also conducted a review of the available medical evidence at Employer's request and authored a report dated March 7, 2005 summarizing his findings and conclusions. EX 21A. His report notes that the Miner had a prolonged hospitalization of sixty days in 1997 for quadruple coronary artery bypass graft complicated by extensive postoperative pneumonia and respiratory failure requiring mechanical ventilation. *Id*. at 5. A 1999 cardiopulmonary exercise study revealed marked limitation of exercise due to cardiac function but not gas exchange or ventilatory function. *Ibid*. The Miner had recurrent episodes of pneumonia following his artery bypass graft. *Ibid*. Spirometry and blood gas test results from 1986 to 1997 before the coronary artery bypass graft were normal, or nearly normal, indicating the total absence of a restrictive abnormality. *Ibid*. According to Dr. Tuteur:

> Thereafter, particularly after the coronary artery bypass graft and the extraordinarily horrific postoperative course in part complicated by postoperative necrotizing pneumonia, pulmonary function worsened dramatically yet still in 1999 there was no restrictive abnormality again as determined by a normal total lung capacity. As late as November, 1999 arterial blood gas analysis was not only normal at rest, but during exercise – the same exercise that was limited because of myocardial (left ventricular) dysfunction. . . .
>
> . . . . .
>
> Following the continued complicated downhill course characteristic of progressive ischemic cardiomyopathy superimposed on severe post-inflammatory changes within the lung and recurrent pneumonias as well as acute on top of chronic congestive heart failure further complicated by pulmonary hypertension secondary to left ventricular dysfunction, Mr. Burns died at the age of seventy-nine years. An autopsy was performed. Six pathologists reviewed the autopsy material and all agreed that pathologic criteria for the diagnosis of coal workers' pneumoconiosis were fulfilled; however, coal workers' pneumoconiosis was considered mild by most and of insufficient severity and profusion to cause clinical symptoms, physiologic impairment, disability, or contribute to death. All reviewers identified the extensive pneumonia, pulmonary artery thickening, and congestive heart failure. On gross examination, no pulmonary emboli were observed.

Id. at 5-6.

Dr. Tuteur concluded the Miner had simple coal workers' pneumoconiosis which was of insufficient severity and profusion to produce clinical symptoms, physical examination abnormalities, impairment of pulmonary function, or even significant radiographic change. *Id*. at 13. He further concluded that the Miner's most significant health problem was his coronary artery disease, and that following his artery bypass graft surgery he suffered "a multiplicity of serious and prolonged complications including necrotizing pneumonia resulting in the development of significant impairment of pulmonary function including the susceptibility for more infections." *Ibid*. According to Dr. Tuteur, "the clinical course spiraled downward with intractable congestive heart failure, debilitating dysrhythmias, recurrent pneumonias, disruption of diabetic control, advancing peripheral vascular disease including carotid artery disease, stroke, and death." *Ibid*. Dr. Tuteur opined that neither coal workers' pneumoconiosis nor any other coal mine dust-induced disease process produced disability during the Miner's life or contributed to, caused, or hastened his death. *Ibid*.

Dr. Tuteur criticized Dr. Perper's conclusion that the Miner suffered from a combined obstructive and restrictive ventilatory defect, noting that all measurements of total lung capacity in the Miner's case were normal and reduced total lung capacity is [a]n absolute criteria for the diagnosis of restrictive abnormality . . . ." *Id*. at 13. He also disputed Dr. Perper's conclusion that the autopsy demonstrated significant and substantial coal workers' pneumoconiosis, noting that the clear consensus among pathologists was that, although the criteria for the diagnosis of simple coal workers' pneumoconiosis were met, the extent and profusion of the changes found were very mild and of insufficient severity and profusion to produce clinical symptoms or impairment of function. *Ibid*.

Dr. Tuteur also criticized Dr. Oesterling's conclusions, stating "his report tends to equate centrilobular emphysema with focal emphysema in coal workers' pneumoconiosis." According to Dr. Tuteur, relevant American medical literature recognizes that focal emphysema differs from centrilobular emphysema in that centrilobular emphysema tends to be a destructive phenomenon due to necrotizing pneumonias and/or chronic inhalation of tobacco smoke. *Id*. at 14.

Dr. Tuteur concluded, based on all the evidence, that the Miner had simple, but not complicated, coal workers' pneumoconiosis at the time of death, that he had no pulmonary impairment when he retired from mining in 1986, and that the subsequent impairment "was due to a combination of thoracic trauma associated with an automobile accident and the consequences of surgical treatment and postoperative morbidity following coronary artery bypass graft and aortic valve replacement." *Id*. at 14. It was his opinion that the Miner did not have any pulmonary disease caused by his coal mining employment that rendered him totally disabled before death, and that the Miner did not die as a result of coal workers' pneumoconiosis or any other coal mine dust-induced disease process. *Ibid*.

Dr. B.T. Westerfield

Dr. Westerfield conducted a review of the available medical evidence at Employer's request and concluded in a report dated September 26, 2001 that the Miner had simple coal workers' pneumoconiosis, category 1/1, based on chest x-rays. DX 34C. He further concluded that the Miner had moderate to severe restrictive lung disease which was not the result of coal workers' pneumoconiosis. He wrote:

> Mr. Burns' pulmonary impairment is due primarily to complications of heart valve replacement surgery and a motor vehicle accident. Mr. Burns had multiple rib fractures, pneumothorax and lung contusion from an auto accident in 1989. This left scarring on his lungs. The more serious injury to his lungs was as complication of aortic valve replacement in 1997. He developed severe pulmonary complications including bilateral pleural effusions and respiratory distress syndrome requiring mechanical ventilation. Convalescence was prolonged and he was hospitalized for two months. He was left with severe scarring of lung parenchyma and pleura which resulted in restrictive lung disease. Coal Workers' Pneumoconiosis has no connection to this condition and he has no impairment as a result of inhalation of coal dust. Infact, [sic] in 1986, prior to any of these events, Mr. Burns had normal pulmonary function.

Ibid. (bolding and underlining omitted). Dr. Westerfield opined that the Miner was totally and permanently disabled from a respiratory disability which was due to cardiac surgery and chest trauma from a motor vehicle accident.

Dr. Erika C. Crouch

Dr. Crouch reviewed the autopsy and biopsy evidence at Employer's request and prepared a pulmonary pathology consultation report dated February 12, 2003. EX 21B. With respect to her microscopic evaluation of the five autopsy slides she reviewed, she noted, *inter alia*, they revealed "numerous abnormalities" including areas of organizing bronchopneumonia superimposed on emphysema with mixed centriacinar, panacinar, and distal acinar patterns, as well as scattered coal dust macules characterized by collections of coal dust containing macrophages, some of which were associated with focal emphysema. Dr. Crouch noted the presence of a dust micronodule but no dust nodules or larger lesions of massive fibrosis or complicated pneumoconiosis. Microscopic examination also showed some dust-related lesions associated with dense fibrous tissue suggestive of inhalation of coal dust containing crystalline silica, and moderate numbers of short needle-like particles consistent with silicates. Dr. Crouch's diagnoses included simple coal workers' pneumoconiosis, organizing pneumonia, necrotizing granulomatous inflammation, emphysema (mixed patterns), and vascular changes consistent with pulmonary hypertension. Under the heading "Comment," she wrote:

> The lungs show coal dust deposition with histologic changes of simple coal workers' pneumoconiosis characterized by scattered coal dust macules and a micronodule. No dust nodules or lesions of progressive massive fibrosis or complicated pneumoconiosis are observed. Consistent with these findings, the

> autopsy report describes does not identify [sic] parenchymal nodules other than the lower lobe area of consolidation consistent with pneumonia. Given the mixed patterns of emphysema, the major risk factor for alveolar destruction was cigarette smoking. The extensive pleural adhesions described in the autopsy report are not attributable to coal dust and could reflect prior episodes of pneumonia and/or the effects of prior thoracic trauma, which require chest tube placement. The etiology of the pulmonary vascular changes is uncertain but most likely reflects some combination of this patient's cardiac and obstructive lung disease. The dust-related disease is of insufficient severity to have caused these changes. Thus, coal dust deposition could not have caused a clinically significant degree of functional impairment or disability and could not have caused, contributed to, or otherwise hasten [sic] this patient [sic] death.

EX 21B at 2.

Dr. Richard L. Naeye

At Employer's request, Dr. Naeye reviewed the available medical records, as well as biopsy and autopsy slides, and reported his findings and conclusions in a report dated April 20, 2003. DX 58. He summarized his conclusions in this case as follows:

> The minimum findings required to make the diagnosis of simple coal workers' pneumoconiosis (CWP) are present in the lungs of Thomas Burns. They include several anthracotic macules with a small amount of associated fibrosis in the lung tissues removed for analysis in 1996. The size and extent of these lesions are far too small for them to have had any measurable effect on lung function, so they could not have caused any disability or contributed in any way to this man's later death. In 1997 cardiac aortic valve surgery led to scarring of lung parenchyma with resultant restrictive lung disease in a localized area of one lung. High resolution CT scan images showed "extensive post-inflammatory scarring, both within the lung parenchyma and the pleura". Most important, high resolution cuts of the upper lung fields showed no findings of coal workers' pneumoconiosis. This is important because CWP lesions characteristically develop in the upper lung lobes before the lower lobes. In the present case the lesions were in the lower lobes of Thomas Burns' lungs.
>
> The lung tissues removed at autopsy for microscopic review have anthracotic lesions with associated black pigment. Careful inspection strongly suggests that the association is not the results of toxic products in coal mine dust. . . .
> . . . .
>
> Finally, the results of this man's pulmonary function studies over the years is characteristic of many ex-coal miners who smoked cigarettes in their earlier life, developed chronic bronchitis as well as slowly progressive centrilobular emphysema which eventually led to disability and sometimes to death. Cigarette smoking makes a several-fold greater contribution to the genesis of these latter

> two disorders than does prolonged exposure to coal mine dust. Thomas Burns reported he smoked up to a pack of cigarettes/day for 17 years, quitting in 1958. Among eastern US bituminous coal miners who worked underground 40 or more years the predicted prevalence of simple CWP at a respirable coal mine dust level of 2 mg/m was 102 for smokers vs. 22/1000 for non-smokers. The respective values were 87 and 18 at a respirable coal mine dust level of 1 mg/m_3. Thomas Burns worked under ground for only 10 years. From pulmonary function studies he had a record of fluctuating chronic bronchitis with slowly progressive and eventually disabling centrilobular emphysema.

Id. at 2-3. Dr. Naeye concluded that the Miner had simple coal workers' pneumoconiosis which was never severe enough to cause disability, and that cigarette smoking was the dominant cause of his chronic bronchitis, progressive centrilobular emphysema, and death. *Id*. at 3. He also noted that the damage done to the microcirculation in his heart by smoking contributed to his disability and death. *Ibid*.

In a supplemental report authored by Dr. Naeye on December 4, 2003, he wrote that he had reviewed Dr. Perper's August 20, 2003 report and that they had both reviewed the same clinical information in this case. EX 12. Dr. Perper, according to Dr. Naeye, is a forensic pathologist who has a good record of publications in forensic pathology but no record of research or publications in the field of coal workers' pneumoconiosis. *Id*. at 1. In contrast, Dr. Naeye has authored multiple research papers and book chapters dealing with coal workers' pneumoconiosis. *Ibid*.

Dr. Naeye disagreed with Dr. Perper's conclusion that the Miner had clinically significant lung disease when he died based, in part, on the normal pulmonary function and blood gas tests performed shortly after the Miner retired and later studies documenting severe abnormalities in lung function. *Ibid*. Dr. Naeye further stated that cigarette smoking makes a several-fold greater contribution to the genesis of centrilobular emphysema and chronic bronchitis than does prolonged exposure to coal mine dust. *Ibid*. He wrote: "Once established by cigarette smoking centrilobular emphysema progresses, even when miners and ex-miners stop smoking." *Ibid*. Dr. Naeye also asserted that Dr. Perper misinterpreted the nature and clinical significance of the black pigment and birefringent crystals seen in the slides, noting that the black pigment "is in an amorphous form which is non-toxic and therefore had no effect on lung function." *Id*. at 1-2. He further noted that the black pigment in the Miner's lungs did not uniformly have fibrosis associated with it, and almost all of the birefringent crystals mixed with the black pigment were far too large to have had toxic potential. *Id*. at 2. While Dr. Naeye agreed with Dr. Perper that there was a large amount of black pigment present in some areas of the Miner's lung tissue, he stated that the fact there was black pigment in some lymph nodes which had very little associated fibrosis was important since the tiny birefringent crystals that are responsible for fibrosis in coal workers' pneumoconiosis continuously migrate in lung tissue but remain much longer, if not permanently, in lymph nodes. *Ibid*. He wrote: "Thus, when fibrosis in the current case is more prominent and advanced in lung tissues than in lymph nodes the likelihood is that most of the fibrosis in the lung tissues had a non-occupational origin." *Ibid*.

Dr. Naeye also stated that the points made in his original report remained valid, including his conclusion that the distribution of lesions noted on the Miner's chest x-rays are atypical for coal workers' pneumoconiosis. *Id*. at 2. According to Dr. Naeye, Dr. Perper "places far too much weight on his quantitative assessments of lesions in the lung tissues available for microscopic review." It is common, Dr. Naeye stated, for autopsy prosectors to "take the most damaged lung tissues they can find for microscopic review because this is the routine practice of pathologists who day in and day out are called upon to make diagnoses, not quantitative assessments for legal purposes." *Ibid*. It is therefore important in assessing coal workers' pneumoconiosis, he stated, to look at chest x-ray and pulmonary function tests. *Ibid*. Dr. Naeye concluded that the Miner's lung function decreased after he quit mining coal because of damage processes set in motion by his earlier cigarette smoking. *Ibid*.

Finally, Dr. Naeye wrote that "surface coal mining only very rarely produces CWP lesions that have any clinical significance . . . [and] they are almost always the consequence of drilling to remove the hard rock overburden over coal seams." The Miner's records, according to Dr. Naeye, revealed no such prior work.

Dr. Joshua A. Perper

Dr. Perper reviewed the available medical evidence at Claimant's request and authored a report dated August 20, 2003 in which he set forth his findings and conclusions. DX 68.[5] Dr. Perper criticized the report of Dr. Tuteur concerning his October 13, 2000 examination of the miner, noting, in part, that Dr. Tuteur incorrectly stated in his report that there was no radiological or clinical evidence of pneumoconiosis despite the fact that such evidence was found by qualified radiologists and clinicians. *Id*. at 12. Dr. Perper also concluded that the September 26, 2001 report of Dr. Westerfield was "severely flawed and incorrect" because, *inter alia*, it failed to mention the Miner's 40 year exposure to coal dust, omitted chest x-ray interpretations by highly qualified physicians showing complicated pneumoconiosis, and omitted evidence from pulmonary function tests indicating both restrictive and obstructive pulmonary defect. *Id*. at 14-15. Similarly, Dr. Perper found the April 20, 2003 report of Dr. Naeye "unbalanced and incorrect and his conclusions flawed" because, Dr. Naeye failed to note respiratory symptoms other than shortness of breath, such as cough, expectoration of mucus and hemoptysis, ignored chest x-ray evidence of both simple and complicated coal workers' pneumoconiosis, and improperly attributed the Miner's restrictive pulmonary defect to chest trauma while ignoring the fact that it was clearly evident before its occurrence. *Id*. at 21. Dr. Perper also asserted that Dr. Naeye minimized the severity and extent of microscopic pathological findings of coal workers' pneumoconiosis, and his opinion that coal workers' pneumoconiosis did not contribute to or hasten the Miner's death was flawed for a variety of reasons. *Id*. at 22-25

Based on his review of the autopsy slides, Dr. Perper diagnosed: (1) simple coal workers' pneumoconiosis, moderate to severe, with micro-nodules and macronodules, mixed coal dust and

[5] In her letter of September 16, 2005 which accompanied Claimant's original post-hearing brief, Claimant's counsel questioned whether DX 68 included Dr. Perper's original medical report, color photographs, and resume. It does. References to Dr. Perper's report in this decision are to the typewritten page number at the bottom of the report rather than the handwritten page numbers appearing thereon.

silicotic types, and birefringent silica crystals; (2) moderate to severe centrilobular emphysema; (3) sclerosis of intra-pulmonary blood vessels consistent with pulmonary hypertension and cor pulmonale; and (4) acute bronchopneumonia. *Id*. at 27.

Based on all the medical evidence, Dr. Perper concluded that the Miner had substantial and significant coal workers' pneumoconiosis. *Id*. at 30-31. He further concluded that the Miner suffered from centrilobular emphysema caused by exposure to mixed coal dust containing silica and coal workers' pneumoconiosis. *Id*. at 31-32. Dr. Perper also found that the Miner's pulmonary impairment was totally disabling, and coal workers' pneumoconiosis was a substantial contributing cause of his disability. *Id*. at 33-34. It was also Dr. Perper's opinion that the Miner's coal workers' pneumoconiosis was a significant and primary contributory cause of, and hastening factor in, his death. *Id*. at 35-36.

On March 23, 2005, Dr. Perper authored a supplemental report in which he addressed the opinions of Drs. Naeye, Caffrey, Oesterling, Tuteur and Repsher. CX 8. With respect to Dr. Naeye's criticism of his original report, Dr. Perper noted, *inter alia*, that a large body of medical literature, as well as NIOSH and the Department of Labor, recognize that occupational exposure to coal mine dust results in centrilobular emphysema and its corresponding chronic obstructive pulmonary disease beyond any effects of cigarette smoking. *Id*. at 3. He further asserted that, contrary to Dr. Naeye's statements, centrilobular emphysema does not progress after cessation of smoking, and both silica crystals and silicates are fibrogenic. Dr. Perper also took issue with Dr. Naeye's conclusion that fibrosis in the Miner's lung tissue had a non-occupational origin because it was more prominent there than in the Miner's lymph nodes. He noted in part that severe fibrosis of the pulmonary lymph nodes occurs in some cases only after the ability of the lung to accommodate coal dust containing silica has been exceeded and the severity of fibrosis in the pulmonary lymph nodes depends on the airborne concentration of mixed coal dust and on individual variations in lymphatic drainage efficiency. *Id*. at 3-4.

With respect to Dr. Caffrey's December 29, 2003 medical report, Dr. Perper noted that Dr. Caffrey diagnosed moderate simple coal workers' pneumoconiosis which may result in pulmonary dysfunction and cause or hasten death. *Id*. at 7. He further asserted that Dr. Caffrey's opinion on whether coal workers' pneumoconiosis contributed to or hastened the Miner's death was "unclear and vacillating with statements which are confusing and/or contradictory . . . ." *Ibid*. Dr. Perper also addressed Dr. Caffrey's various criticisms of his earlier report. *Id*. at 8-9.

With respect to Dr. Oesterling's pathology report, Dr. Perper found it "particularly flawed" for a variety of reasons including failing to list the "extensive medical records" which Dr. Oesterling claimed to have reviewed, and failing to summarize or discuss important clinical findings. *Id*. at 10. Dr. Perper took issue with Dr. Oesterling's criticism of Dr. Perper's findings regarding pneumoconiotic lesions based on the presence of anthracotic pigment. *Id*. at 10-11. According to Dr. Perper, Dr. Oesterling's claim that this pigment was hemosiderin (a pigment derived from hemoglobin) was contradicted by the fact than none of the five pathologists who microscopically examined the autopsy lung sections other than Dr. Oesterling diagnosed the presence of hemosiderosis. *Id*. at 11.

Dr. Perper described Dr. Repsher's April 12, 2004 report as somewhat contradictory in that he acknowledged the existence of pathological evidence of simple coal workers' pneumoconiosis but stated there was "only controversial radiological evidence" of the condition. *Id*. at 14. According to Dr. Perper, pathological evidence is "clearly the diagnostic gold yardstick for diagnosing coal workers pneumoconiosis, and not the controversial radiological shadows." *Ibid*. He further asserted that Dr. Repsher was "definitely incorrect" in stating that, although the inhalation of coal mine dust may cause COPD, the average impairment is so small as not to be discernable in an individual miner. Dr. Perper cited various authorities, including NIOSH and the Department of Labor, as recognizing that exposure to mixed coal dust and coal workers' pneumoconiosis can result in centrilobular emphysema manifested by substantial pulmonary disability and contributing to or hastening death. *Ibid*. Dr. Perper also disputed Dr. Repsher's conclusion that the Miner's respiratory symptoms were due exclusively to congestive heart failure since shortness of breath and other symptoms and findings occurred when the Miner was not in cardiac failure. *Id*. at 15. Finally, Dr. Perper took issue with Dr. Repsher's conclusions that the Miner's ischemic cardiomyopathy was the sole cause of death and his mild to moderate ventilatory impairment was due mostly to his heart failure and was not the result of exposure to coal mine dust. *Ibid*. According to Dr. Perper, "Dr. Repsher unreasonably excludes any role in the causation of death for the miner's substantial simple coal workers' pneumoconiosis and the complicating COPD and pneumonia."

With respect to Dr. Tuteur's March 7, 2005 report, Dr. Perper disputed Dr. Tuteur's conclusion that there was "a clear consensus" among pathologists that the Miner's coal workers' pneumoconiosis was very mild and insignificant from a clinical viewpoint, noting the results reported by Drs. O'Neill, Naeye, Caffrey, and Oesterling which he believed showed significant and substantial coal workers' pneumoconiosis. *Id*. at 16-17. Dr. Perper also defended his opinion that the Miner had both restrictive and obstructive lung disease, noting that the medical records showed restrictive lung disease and the autopsy findings showed evidence of obstructive lung disease. *Id*. at 17. Finally, Dr. Perper argued that Dr. Tuteur unreasonably excluded the Miner's substantial coal workers' pneumoconiosis and causally associated COPD and pneumonia from any role in the causation of death. *Ibid*.

In conclusion Dr. Perper stated that the findings and conclusions set forth in his August 20, 2003 report had not changed based on his review of the medical opinion evidence obtained by Employer. *Id*. at 17. It remained his opinion that the Miner's more than 40 years of occupational exposure to coal mine dust caused significant, substantial and severe simple coal workers' pneumoconiosis, which was causally associated with his centrilobular emphysema, and that his respiratory condition was a substantial contributory cause of death. *Id*. at 17, 21.

Dr. Robert A.C. Cohen

Dr. Cohen reviewed the available medical evidence at Claimant's request and authored a report dated December 9, 2003. CX 5. He opined, *inter alia*, that: the Miner had symptoms consistent with chronic lung disease beginning in the 1980's; pulmonary function testing performed since 1997 demonstrated a worsening of his obstructive lung disease and showed a severe obstructive defect "consistent with his 40 years exposure to coal dust and probably not at all to his remote and modest smoking history;" x-ray evidence supported a diagnosis of

pneumoconiosis which was confirmed by autopsy evidence which revealed lesions of complicated pneumoconiosis; and the Miner had no history of any other occupational exposure which could cause his coal workers' pneumoconiosis or obstructive lung disease. *Id*. at 22-23. Dr. Cohen wrote that pulmonary function testing results revealed a severe obstructive lung disease which precluded the Miner from engaging in the physical exertion required of his coal mine employment. *Id*. at 23. He further noted:

> It is my opinion, within a reasonable degree of medical certainty, that Mr. Burns' long-term exposure to coal dust was a primary cause of his pulmonary disability as manifested by his restrictive and severe obstructive defect with diffusion impairment and gas exchange abnormalities. . . .

Ibid. Dr. Cohen also concluded that the Miner's severe respiratory impairment was "definitely a result of his coal dust exposures and not his remote and limited smoking history." *Id*. at 27. He criticized Dr. Naeye's belief that smoking contributes much more to obstructive lung disease than does occupational exposure, noted that Dr. Naeye cited no authority for his opinion, and wrote that relevant medical literature "shows that coal dust exposure contributes almost as much as smoking does to obstruction." *Ibid*. Dr. Cohen noted that the Miner smoked for only 15-17 years, stopped smoking 13 years after he started mining, and continued mining employment for another 27 years thereafter with no pulmonary condition or impairment until more than two decades after he stopped smoking.[6] *Ibid*. He also wrote: "There is no authority for the proposition that lung damage occurring 28 years after cessation relates to a remote and limited smoking history." *Ibid*.

With respect to Drs. Tuteur's and Westerfield's partial attribution of the Miner's pulmonary problems to his 1989 motor vehicle accident, Dr. Cohen found those opinions speculative since neither physician had "medical records related to the accident which would permit a conclusion or even an impression that the resulting injuries produced pulmonary problems." *Id*. at 27. Dr. Cohen further noted that the Miner had shortness of breath as early as 1985, four years before the accident, and trauma associated with an automobile accident would result in lung damage that would appear on pulmonary function tests as restrictive in nature due to fractures or pleural disease. *Ibid*. According to Dr. Cohen, the Miner's pulmonary impairment was "primarily obstructive in nature, which could only be caused by his exposure to coal mine dust and his remote and very modest exposure to tobacco smoke." *Id*. at 28.

Dr. Cohen also disagreed with Drs. Tuteur and Westerfield to the extent they attributed any of the Miner's pulmonary problems to heart disease based on his 1997 surgery for coronary artery bypass and heart valve replacement. He noted that the records of Dr. Crabtree, the Miner's pulmonary specialist, and Dr. Moses, his cardiologist, reflected that the Miner recovered completely from the surgery although convalescence was lengthy and difficult. Dr. Cohen reiterated that the Miner had shortness of breath as early as 1985, well before the heart surgery,

[6] Dr. Cohen, like Dr. Perper, appears to have overestimated the number of years Mr. Burns smoked and underestimated the length of time between when the Miner quit smoking and when he first developed a pulmonary impairment. Any error in this regard, however, would clearly not change his opinion regarding the lack of any association between Mr. Burns remote smoking history and his development of a pulmonary condition.

and stated that heart disease would not have caused the Miner's obstructive pulmonary impairment since heart disease could result only in restrictive impairment. *Id*. at 28.

Dr. Cohen further disputed Dr. Tuteur's partial attribution of the Miner's pulmonary problems to an endobronchial inflammatory process noting that an October 6, 1999 fiberoptic bronchoscopy performed for evaluation of possible hemoptysis revealed no endobronchial tumor. *Id*. at 29. According to Dr. Cohen, endobronchial inflammation is a non-specific diagnosis with many causes, and Dr. Tuteur neither explained why he made this diagnosis nor what the cause of it was if it was present in the Miner. Dr. Cohen wrote: "It is clear that Mr. Burn's [sic] was exposed to coal mine dust which can cause chronic bronchitis and airways inflammation." *Ibid*.

Dr. Cohen also opined that the Miner's pulmonary condition declined significantly since 1987 based on significant positive x-ray evidence after 1987, pulmonary function studies showing severe reduction in FVC, FEV1, MVV and diffusion capacity, and worsening gas exchange shown by the results of blood gas studies. *Id*. at 29-30. Dr. Cohen further noted that the Miner's clinical symptoms and exercise intolerance also reflected worsening impairment.

Finally, Dr. Cohen concluded that the Miner died as a result of his pulmonary condition. He wrote:

> Mr. Burns had severe simple pneumoconiosis as well as severe obstructive lung disease due to his coal mine dust exposure. He had severe obstruction and diffusion impairment and gas exchange impairment near the time of death, an indication of substantial and clinically significant interstitial and obstructive lung disease. I have no doubt that his coal mine dust exposure was a primary cause or substantial contribution to the development of his severe impairment, his medical pneumoconiosis and his legal pneumoconiosis, all of which significantly contributed to his respiratory death.

Id. at 30.

Dr. Cohen concluded, based on his review of all the available medical evidence, that:

> [t]he sum of the medical evidence in conjunction with [the Miner's] work history indicates that [his] 40 years of coal dust exposure was a significantly contributory cause of his severe obstructive lung disease, diffusion impairment, and gas exchange abnormalities on resting and exercise blood gases which caused him to be disabled and hastened his death from pneumonia.

Id. at 30.

Dr. P. Raphael Caffrey

Dr. Caffrey reviewed the available medical evidence and surgical pathology slides at Employer's request and authored a December 29, 2003 report in which he set forth his findings

and conclusions. EX 13. Dr. Caffrey opined that the Miner suffered from a moderate degree of simple, but not complicated, coal workers' pneumoconiosis. *Id*. at 6. In response to being asked whether the Miner was totally disabled as a result of any pulmonary condition caused by his coal mining experience, Dr. Caffrey wrote:

> It is I believe extremely difficult to separate his cardiac condition which was due to severe coronary artery atherosclerosis (which was accelerated by diabetes mellitus) and his CWP as relates to his pulmonary condition. Mr. Burns' severe coronary artery atherosclerosis, with the complications thereof requiring cardiac surgery, as well as bilateral carotid endarterectomies, had nothing to do with his employment in the coal mining industry. . . . Definitely prior to his death Mr. Burns was totally disabled from his cardiac disease and his respiratory disease, but certainly he would not have been able to work anyway because he was 78 years of age. It should also be noted that the pulmonary problems Mr. Burns suffered from were certainly accelerated following his motor vehicle accident in 1989 when he received trauma to the right thorax with multiple rib fractures and developed pleural adhesions and fibrosis. . . .

Id. at 7. As far as cause of death, Dr. Caffrey concluded that "the immediate cause of death was acute bronchopneumonia with abscess formation in an individual who had cardiomyopathy, congestive heart failure, COPD, and CWP." *Ibid*. He noted that the Miner had a variety of significant medical diseases which were not related to his coal mine employment, one of which was centrilobular emphysema which he noted "some of which may be due to simple CWP . . . ." *Ibid*. Dr. Caffrey further noted, however, that it did not appear the bronchitis or centrilobular emphysema were "debilitating" irrespective of the cause.

When asked whether the Miner's death was caused or hastened by any condition associated with his coal mining, Dr. Caffrey wrote that he did not believe "there is any objective way to prove or disprove an opinion of 'yes' or 'no' to that question." *Id*. at 8. He went on to state that he "definitely" believed that "if Mr. Burns only had a moderate degree of simple CWP he would certainly not have been disabled as he was and the probability of his dying at this time, even though he was 79 years of age is unlikely." *Ibid*. The atherosclerotic conditions the Miner had, according to Dr. Caffrey, were unrelated to his coal mine employment and "could have caused and definitely hastened his death." *Ibid*.

Dr. Caffrey went on to list opinions of Dr. Perper with which he disagreed, including Dr. Perper's conclusion that the Miner was permanently and totally disabled as a result of his severe coal workers' pneumoconiosis and associated centrilobular emphysema. *Id*. at 8. Dr. Perper, according to Dr. Caffrey, ignored or overlooked the severe cardiac problems suffered by the Miner in reaching this conclusion. These problems, he wrote, "could have caused . . . the patient's severe pulmonary problems, whether they alone did cause his severe pulmonary problems I certainly cannot objectively say." *Ibid*. He went on to note that he did not believe the Miner's centrilobular emphysema played a role in causing or contributing to his death or in causing any significant pulmonary disability prior to death. *Id*. at 8-9.

Dr. Everett F. Oesterling

Dr. Oesterling reviewed the available medical evidence at Employer's request and authored a report dated February 4, 2004 in which he set forth his findings and conclusions. EX 14. His report summarizes his histologic findings as follows:

1. There is evidence of severe passive pulmonary congestion secondary to a failing left ventricle.
2. With the passive congestion there has been extensive intra-alveolar hemorrhage resulting in the release of prominent quantities of hemosiderin.
3. Hemosiderin as an irritant has produced fibrosis in the lower lobes and right middle lobe of the lunges, i.e., hemosiderosis.
4. There are modest quantities of black pigment mixed with the prominent hemosiderin in some areas, particularly the pleural surfaces within the lower lobe.
5. There is evidence of extensive pneumonia within the lower lobes due to opportunistic microorganisms complicating the passive congestion.
6. The biopsy specimens demonstrated evidence of passive congestion in 1996.
7. The lymph nodes obtained during the surgical procedure in 1996 showed limited evidence of mine dust exposure and reaction to the same.
8. Based on the sum total there is sufficient change present for a diagnosis of mild micronodular coalworkers' pneumoconiosis.
9. This level of change is insufficient to have altered pulmonary function.
10. Without alterations in function no lifetime disability would have resulted from coalworkers' pneumoconiosis.
11. With the limited structural change there would have been no contribution to death, thus the exposure did not hasten, contribute to, or cause this gentleman's demise.
12. Lifetime respiratory disability is due to central progressing to panlobular pulmonary emphysema which is unrelated to his dust exposure.

Id. at 6-7. Dr. Oesterling concluded that smoking was the cause of the Miner's emphysema and that his vascular disease was unrelated to coal mine dust exposure. *Id*. at 7. He also noted various chest x-ray reports referenced fibrotic changes in both lung bases which he stated were atypical of the normal distribution of coal workers' pneumoconiosis. *Id*. at 7-8. Dr. Oesterling wrote:

> Hopefully the photomicrographs have clearly shown the confusion that has arisen in this case due to two pigments, hemosiderin and anthracotic pigment. The predominate fibrotic response throughout his lung sections is in response to hemosiderin and not mine dust. . . .

Id. at 8. Dr. Oesterling noted the absence of any reference to hemosiderin in the portion of Dr. Perper's August 20, 2003 report discussing his review of the autopsy slides, and viewed this

omission as "an effort [by Dr. Perper] to concentrate on mine dust and ignore the primary disease process which was present." *Ibid*. Dr. Oesterling also rejected Dr. Perper's exceptions to the opinions of Dr. Naeye, finding that Dr. Naeye's "conclusions are not dissimilar to mine and are far more accurate than those set forth within Dr. Perper's report." *Id*. at 9.

Dr. Oesterling was deposed in this matter on March 10, 2005 and reiterated the bases for his findings and conclusions. DX 22.

Dr. Lawrence Repsher

Dr. Repsher reviewed the available medical evidence at Employer's request and authored a report dated April 12, 2004. EX 17. Based on his review of the evidence, he concluded that the Miner "had simple histologic coal workers pneumoconiosis, but he did not suffer from during his life nor did he die, as a result of any pulmonary respiratory condition, either caused by or aggravated by his employment in the coal mining industry." *Id*. at 15. Dr. Repsher noted that there was pathological evidence of simple coal workers' pneumoconiosis, but "only controversial radiologic evidence to support the diagnosis of coal workers pneumoconiosis." *Ibid*. He also noted that the Miner had a mild to moderately severe obstructive ventilatory impairment due to chronic bronchitis, recurrent pneumonitis, possible bronchiectasis, and pulmonary emphysema, none of which were due to coal dust exposure. *Ibid*. He opined that, prior to his death, the Miner had a reduced capacity to perform his coal mine employment because of his respiratory impairment, but exposure to coal mine dust had no discernible effect on the Miner's clinical course prior to his death. *Ibid*. He also concluded that coal workers' pneumoconiosis did not cause, contribute to, or in any way hasten the Miner's death. *Ibid*.

With regard to specific medical evidence he reviewed, Dr. Repsher wrote that the Miner's cardiology records revealed progressive deterioration of his cardiovascular function prior to and following his quadruple coronary artery bypass grafting and aortic valve replacement. *Id*. at 5. Those records further showed recurrent episodes of severe congestive heart failure occurred, and became more frequent, starting in 2001 and terminating with the Miner's death in 2002. The Miner's episodes of congestive heart failure were worsened by repeated episodes of pneumonia, and it was Dr. Repsher's opinion that cardiovascular compromise with severe biventricular decompensation accounted for essentially all of the Miner's progressive disability.

Dr. Repsher disagreed with Dr. Crabtree's post mortem consultation report of October 29, 2002 in which he stated that the Miner had a history of severe, end-stage pneumoconiosis, significant bronchiectasis, chronic pulmonary fibrosis, and severe end-stage disease. *Id*. at 6. According to Dr. Repsher, spirometry results following treatment of congestive heart failure and pneumonia did not demonstrate evidence of severe obstructive ventilatory impairment, and arterial blood gases showed only mild arterial hypoxemia as of March 1, 2002. He further noted that CT scans of the chest did not demonstrate the presence of bronchiectasis or evidence of significant coal workers' pneumoconiosis.

Dr. Repsher also disagreed with Dr. Migone, the physician who signed the Miner's death certificate, inasmuch as she attributed the cause of death to right lower lobe pneumonia and

ascribed the pneumonia to the Miner's pneumoconiosis. *Id*. at 6. He wrote: "Coal workers pneumoconiosis does not cause pneumonia and does not cause congestive heart failure or lead to a cardiopulmonary arrest." *Ibid*.

Dr. Repsher also disagreed with Dr. Cohen's evaluation of the pulmonary function data. *Id*. at 13. He noted that the October 26, 2000 spirogram revealed values which "comfortably exceed the Department of Labor's requirements for disability in coal miners." *Ibid*. With regard to the valid pulmonary function tests conducted between November 4, 1986 and October 26, 2000 which showed reductions in forced vital capacity and forced expiratory flow, he wrote:

> In each instance, these correlate with objective chest x-ray and often chest CT scan evidence of either pneumonia [or] congestive heart failure. Following antibiotic treatment for pneumonia and adequate diuresis with diuretics, the chest x-rays improve as do the pulmonary function studies. There is no adequate evidence of a persistently worsening ventilatory defect that one would expect in progressive coal workers pneumoconiosis. In fact, even though the chest CT scans showed evidence of progressive pulmonary emphysema, the objective pulmonary function studies did not confirm the presence of a severe obstructive ventilatory impairment that exceeded disability requirements for coal workers. Sequential echocardiograms demonstrated progressive worsening in Mr. Burns' ischemic cardiomyopathy. . . . Progressive pulmonary hypertension was evolving with the estimated pulmonary systolic pressure rising from 26 on 15 July 1997 to 85 by 18 January 2000. The pulmonary function data showed only a mild to moderate obstructive ventilatory impairment with normal gas exchange on 23 November 1999 at rest and following exercise. Pulmonary emboli had not been diagnosed to account for the rising pulmonary systolic pressure, although the clinical course with punctuated episodes of hemoptysis, pulmonary infiltrates, and bilateral pleural effusions with densities abutting the pleural surface, which certainly enter in the differential diagnosis as the cause of pulmonary hypertension. . . . [R]ecurrent bouts of left-sided ventricular failure with passive pulmonary congestion most likely accounted for the majority of his pulmonary venous hypertension and the findings of passive congestion at autopsy with hemosiderin-induced fibrosis, as documented by Dr. Oesterling in his pathology report. Since pulmonary emboli were not documented at post mortem, this seems like a reasonable explanation for the evolution of severe pulmonary hypertension. It is a much more likely explanation for the pulmonary hypertension rather than simple coal workers pneumoconiosis or pulmonary emphysema. . . .
>
> . . . .
>
> I, therefore, do not believe that the coal dust to which he was last exposed in 1985 played any role whatsoever in the cause of his terminal pneumonitis, congestive heart failure, or cardiopulmonary arrest.

Id. at 13-14.

Dr. Gregory J. Fino

Dr. Fino reviewed the available medical evidence concerning the Miner at Employer's request and thereafter authored a report dated May 6, 2004 outlining his conclusions. EX 18. He concluded that there was sufficient evidence to demonstrate coal workers' pneumoconiosis pathologically, and noted that the miner had "absolutely normal spirometry" in 1986, one year after he stopped working in the mines. *Id*. at 13. He further noted that lung function studies performed 11 years later showed an obstructive ventilatory abnormality, and stated that "[i]t would be unusual to develop new obstructive lung disease when you leave the mines, if one were going to attribute the obstruction to coal mine dust." *Ibid*. Dr. Fino concluded that the Miner's continued pulmonary problems paralleled his significant coronary artery disease, which was unrelated to his coal mine employment, and noted that his "terminal event appeared to be a pneumonia." *Id*. at 14. He believed that it was even more likely, however, that the Miner "died a cardiac death" given his significant history of coronary artery disease. *Ibid*. According to hospital records, the Miner "suddenly experienced shortness of breath, a change in his overall status and a decrease in his oxygenation" which could not have been caused by any chronic lung disease, including disease caused by inhalation of coal mine dust. *Ibid*. Dr. Fino concluded that the event described in the records "could be an acute heart attack, a large stroke or intracranial hemorrhage, or possibly even a ruptured abdominal aortic aneurysm." *Ibid*. Since the autopsy was limited to the lungs only, Dr. Fino wrote, it was not helpful in determining the cause of the Miner's death. *Ibid*.

Dr. Fino wrote, in conclusion:

> Based on review of all of the medical information and even assuming that coal workers' pneumoconiosis contributed to an obstructive abnormality, it is my opinion that it is speculative, at the least, to say that lung disease or even coal workers' pneumoconiosis was a contributing factor or a hastening cause of this man's death. Suffice it to state that, in my opinion, there is insufficient objective evidence to show that coal mine dust inhalation was a contributing or hastening factor in this man's death.
>
> I do not believe that there was any significant evidence to show a diagnosis of complicated coal workers' pneumoconiosis.
>
> I do not believe that there was sufficient objective evidence to show that this man had a disabling coal mine dust-induced lung disease or that coal mine dust inhalation contributed to disability.
>
> Prior to death, he was obviously disabled but it was due to coronary artery disease and heart dysfunction

Id. at 16-17.

G. Hospital and Treatment Records

The record contains multiple medical records submitted by Claimant and Employer from a variety of sources including the following:

Treatment records from Memorial Hospital of Carbondale dated 2002. DX 58 (EX 5).

Included among these records is a discharge summary dated February 26, 2002 which notes diagnoses of congestive heart failure with mitral and aortic insufficiency, pulmonary hypertension, and peripheral vascular disease with carotid artery disease. *Id*. at 457. The attending physician was Ana Migone. *Ibid*. A consultation report by Dr. Coello during this hospital stay notes diagnoses of congestive heart failure with an ejection fraction of less than 24%, peripheral vascular disease, black lung, mitral valve insufficiency and pulmonary hypertension. *Id*. at 458.

The Miner was also admitted to Memorial Hospital of Carbondale on March 28, 2002 where he was diagnosed by Dr. Ana Migone with, *inter alia*, histories of congestive hear failure with ejection fraction of less than 24%, peripheral vascular disease, black lung disease, and mitral insufficiency. *Id*. at 225.

The Miner was again admitted to Memorial Hospital on April 18, 2002 and discharged April 22, 2002. *Id*. at 106-111. Diagnoses by Drs. Paul Salmon and Ana Migone included congestive heart failure exacerbation, history of chronic obstructive pulmonary disease contributing to congestive heart failure exacerbation, and pneumonia. *Id*. at 106. Treatment for his "[c]hronic obstructive pulmonary disease/black lung" was continuation of Flovent and Serevent inhalers. *Id*. at 111.

A consultation report by Dr. Jeffrey Gibbs dated April 19, 2002 notes that a chest x-ray showed right lower lobe consolidation with bilateral pleural effusions. *Id*. at 134. Dr. Gibbs' assessment was shortness of breath and edema compatible with congestive heart failure, doing well with diuresis, no acute coronary ischemic problem, and known severely impaired ejection fraction. *Ibid*.

An admitting summary prepared by Dr. Peter Somers and Dr. Migone dated June 5, 2002 notes that:

> The patient does have a history of black lung. Over the past few days he has had increase in symptoms with some hemoptysis this morning. . . .

Id. at 8.

A consultation report by Dr. Cesar Coello dated June 6, 2002 notes, *inter alia*, impression of atherosclerotic heart disease, congestive heart failure, recurrent pneumonia, severe chronic obstructive lung disease, and paroxysmal atrial fibrillation. *Id*. at 45.

A discharge summary by Dr. Ana Migone dated June 7, 2002 reflected diagnoses of right middle lobe pneumonia, atrial fibrillation, diabetes, congestive heart failure, and chronic obstructive pulmonary disease with black lung disease. *Id*. at 6. The report further notes:

> On the morning of June 7, 2002, the patient suddenly experienced a decrease in oxygen with shortness of breath and had a sudden change in his medical status. The patient stopped breathing at 2:50 a.m. on June 7, 2002.

Id. at 7.

A preliminary autopsy report prepared by Dr. Patrick O'Neill dated June 7, 2002 notes, *inter alia*:

> My preliminary conclusions are based on history provided and the macroscopic findings at the time of the autopsy which reveal the following:
>
> 1. Bilateral lungs with severe pleural fibrinous adhesions.
> 2. Bilateral lungs with severe anthracotic pigment deposition.
> 3. Right lower lung with consolidation and marked pleural thickening.
> 4. Bilateral lungs with dilated pulmonary arteries.
> 5. Barrel shaped chest, generalized muscle wasting, prominent strap muscles in neck, findings consistent with severe chronic obstructive pulmonary disease.
> 6. 17.0 cm midline chest old surgical scar consistent with clinical history of aortic valve replacement.

Id. at 103.

Treatment records from St. John's Hospital dated 1987-2002. DX 58 (EX 6).

According to a discharge summary dated January 10, 1987 prepared by Dr. Moses, the Miner was admitted to St. John's Hospital on January 8, 1987 and cardiac catheterization was performed the following day. According to Dr. Moses, the Miner had relatively few symptoms and his disease was very diffuse.

A letter dated May 31, 1996 authored by Dr. Moses notes that the Miner had undergone cardiac catheterization by Dr. Joel Schneider earlier in the month and was readmitted on May 28, 1996 for cerebral angiography which demonstrated 95% stenosis a the origin of the left internal carotid artery and 50% stenosis of the right internal carotid artery.

According to a letter dated August 7, 1997 by Dr. Moses, the Miner had been admitted to St. John's Hospital on July 14, 1997 for chest pain and worsening heart failure. Quadruple vessel bypass surgery and aortic valve replacement was performed on July 17, 1997 by Dr. Schneider, after which the Miner had decreased level of consciousness and apparently experienced a grand mal seizure. The Miner gradually improved and was ultimately discharged on August 7, 1997.

Dr. Moses obtained a history and performed a physical examination of the Miner on September 30, 1999. His report notes that the Miner "has a very complex past medical history and represents a high risk." He further notes that he should be seen by a pulmonologist and possibly undergo a bronchoscopy prior to a scheduled hernia operation.

A consultation report for chronic obstructive pulmonary disease and hemoptysis dated October 6, 1999 and prepared by Dr. David Crabtree notes that the Miner had recently experienced shortness of breath, dyspnea on exertion, and episodes of hemoptysis which he attributed to a change in his inhaler regimen. The impression was COPD and hemoptysis with an abnormal chest x-ray.

According to an operative report dated October 6, 1999 prepared by Dr. Crabtree, a bronchoscopy revealed mild erythema in the trachea and right endobronchial, more intense inflammation in the left, "especially the left lower lobe and the left lingular segment." A significant amount of mucopurulent secretions was removed, and "a mucosal abnormality in the left lower lobe with some narrowing and erythema, [with] somewhat irritated appearing mucosa. . . was brushed and then biopsied with forceps." The microscopic pathologic diagnosis from the biopsy showed endobronchial tissue with edema, mild chronic inflammation, and reactive epithelia changes.

A letter dated October 8, 1999 from Dr. H. Weston Moses of Prairie Cardiovascular Consultants, Ltd. notes that Mr. Burns was admitted on October 6, 1999 to St. John's Hospital and underwent a bronchoscopy that date by Dr. Crabtree for hemoptysis. There was left upper lobe infiltrate and atelectasis noted and cytology was negative for cancer. According to Dr. Moses, "the problem appears to be mainly one of bronchiectasis and coal miner's lung." A CAT scan of the chest was obtained and revealed "extensive consolidation with one nondiagnostic nodule . . . ." He also wrote:

> Dr. Crabtree would also like to see the patient in follow-up. The patient has rather severe lung disease and there is a question of a lung nodule that will need attention at some point (number two on the CT scan report). Once again, the preliminary cytology report is that no malignant cells are noted from bronchoscopy, fortunately.

Ibid.

A postoperative report by Dr. Gregory Mishkel dated October 27, 1999 noting that the Miner underwent an arch angiography, four vessel cerebral angiography.

> Treatment records from Prairie Cardiovascular Consultants, Ltd. dated 1987-2002. DX 58 (EX 7).

A letter dated October 8, 1999 by Dr. Moses notes that the Miner was hospitalized between October 6-8, 1999 for a large left inguinal hernia.

Dr. Mishkel's consultation report, dated January 9, 2002, notes an impression of, *inter alia*, severe underlying pulmonary disease "as per Dr. Crabtree," evidence of congestive heart failure with underlying coronary artery disease and known ischemic cardiomyopathy, and peripheral vascular disease.

A cardiology consultation summary dated January 14, 2002 by Dr. Moses notes that the Miner was seen the prior week by his colleague Dr. Greg Mishkel for pneumonia and what appeared to be worsening heart failure. An echocardiogram demonstrated severe biventricular failure and significant pulmonary hypertension. The Miner was seen by Dr. Moses on January 31, 2001 for complaints of chronic shortness of breath. An echocardiogram demonstrates rather severe left ventricular dysfunction and mild to moderate mitral regurgitation.

Dr. Moses saw the Miner on January 18, 2000 and noted, *inter alia*, poor left ventricular function and mild to moderate mitral regurgitation "of no obvious significance . . . ."

Treatment records from DuQuoin Satellite Clinic dated 1998-1999. DX 58 (EX 8).

Treatment notes dated January 17 and 20, 2000 by Dr. Travis reflect assessments of diabetes mellitus, hypertension, hemoptysis, black lung, and COPD.

A treatment note dated August 30, 2000 by Dr. E. Clay Travis notes that the Miner was seen that date for recheck and evaluation after an episode of shortness of breath several days earlier. The assessment was COPD and anxiety.

Other treatment records reflect ongoing treatment for hypertension and diabetes.

Treatment records of Dr. David Crabtree dated 1999-2002. DX 19, 34A, 42, 58 (EX 9).

Treatment records of Dr. Crabtree reflect various diagnoses during the period 1999 through 2002 of congestive heart failure, cardiomyopathy, COPD, pulmonary fibrosis, pleural effusion, bronchiectasis, coronary artery disease, and hemoptysis.

Treatment records from Herrin Hospital dated 1997. DX 58 (EX 10).

These records consist primarily of physical therapy progress reports reflecting 22 treatments between October 6, 1997 and December 1, 1997 following the Miner's aortic valve replacement surgery. The Miner reported significant deconditioning following his surgery and difficulty with prolonged standing, climbing stairs, walking, and lifting or carrying light objects.

Treatment records of Dr. Cesar Coello dated 1997-2002. DX 40.

In a consultation report dated March 1, 2002, Dr. Coello noted impressions of ischemic dilated cardiomyopathy with severe systolic dysfunction, pulmonary hypertension with no evidence of decompensation, status post aortic valve replacement, and history of chronic lung disease.

In a letter dated May 1, 2002, Dr. Coello notes that he saw the Miner in cardiovascular follow-up, and that he was doing "fairly good after his last discharge where he was treated for recurrent peripheral edema." He was able to walk with a walker at home, but used a wheelchair elsewhere. Dr. Coelho's impression included congestive heart failure with severe ischemic cardiomyopathy and severe systolic dysfunction, atherosclerotic heart disease, chronic lung disease on oxygen at home, peripheral vascular disease, and recurrent pneumonias.

Dr. Coello saw the Miner on June 6, 2002 and rendered diagnoses of severe systolic dysfunction with evidence of dilated cardiomyopathy and previous inferior wall myocardial infarction, biatrial enlargement, moderate mitral regurgitation, moderate to severe tricuspid regurgitation, and severe pulmonary hypertension.

Diagnostic Test Results from Treatment Records.

The treatment records submitted by the parties contain multiple diagnostic test results from numerous chest x-rays, CT scans, pulmonary function tests, and arterial blood gas studies, most of which are summarized above. Those not noted above include the following:

X-Ray reports:

Exhibit	Date of X-ray	Physician	Interpretation
DX 58 (EX 7)	1/8/87	Gregg	Heart size and pulmonary vascularity within normal limits; focal area of linear fibrosis or discoid atelectasis lateral aspect left lung base; lungs clear of active infiltrates; moderately prominent anterior osteophytes mid thoracic spine.
DX 58 (EX 6)	5/3/96	Stevens	Bibasilar interstitial pattern probably representing fibrosis; multiple old right rib fractures.
DX 58 (EX 6)	5/4/96	Williams	Atelectasis right base anteriorly; active disease doubted; thoracic configuration with COPD.
DX 58 (EX 6)	7/14/97	Williams	COPD; chronic fibrotic infiltrative changes, probably both in middle lobe and lingular segment of left upper lobe; doubt active pulmonary disease at this time; no significant change since 1996.
DX 58 (EX 6)	7/17/97	Sullivan	Post-op changes with mild bibasilar atelectasis.
DX 58 (EX 6)	7/17/97	Sullivan	Interval development of central vascular congestion; right effusion; post-op changes.
DX 58 (EX 6)	7/18/97	Sullivan	Enlarging right effusion; mild central vascular congestion; basilar atelectasis.
DX 58 (EX 6)	7/23/97	Winders	Opacification in right hemithorax similar to previous exam; increased opacific action in left hemithorax as compared to previous exam; question pleural fluid and pulmonary congestion; interval removal of ET tube.
DX 58 (EX 6)	7/24/97	Winders	Considerable bibasilar pleural fluid; underlying parenchymal infiltrations cannot be excluded because of fluid; right-sided jugular line.
DX 58	7/30/97	Winders	Interval removal of right-sided jugular line;

Exhibit	Date of X-ray	Physician	Interpretation
(EX 6)			considerable bibasilar pleural fluid; new area of linear atelectasis in left mid lung.
DX 58 (EX 6)	8/1/97	Kuhn	Persistent bilateral pleural effusions; persistent bilateral atelectatic streaks; heart not enlarged; post surgical changes evident.
DX 58 (EX 6)	8/4/97	Gleason	Post CABG changes noted; prosthetic valve in aortic position; persistent bilateral pleural effusions with associated bibasilar parenchymal opacity consistent with atelectasis and/or infiltrate; mid and upper lung fields remain clear; heart size cannot be accurately evaluated due to overlying opacity; pulmonary vasculature not significantly congested; multiple old right-sided rib fractures.
DX 58 (EX 6)	8/5/97	Stevens	Bilateral residual pleural effusions; no evidence for post procedural complication; no pneumothorax; old right rib fractures.
DX 58 (EX 6)	10/5/99	Gregg	Operative changes of previous CABG; heart size borderline enlarged; no pulmonary vascular congestion; blunting of costophrenic angles suggesting small pleural effusions; some infiltrate or atelectasis in left mid lung; no pneumothorax; mild osteoporosis.
DX 58 (EX 6)	10/8/99	Oncay	S/p median sternotomy and CABG; left pleural effusion; normal cardiac and mediastinal silhouettes; cannot exclude retrocardiac mass density or infiltrate based on single portable film; patchy areas of atelectasis in left mid lung and right mid lung; multiple remote rib fractures along posterolateral aspect of chest involving right ribs.
DX 34A	10/12/99	Russo	Interval development of abnormal opacity in mid to right lower lung compared to 5/11/99 x-ray which is suspicious for pneumonia; increased interstitial markings right lung compared with previous x-ray which may be part of same inflammatory process; could also represent lymphangitic spread of carcinoma in appropriate clinical context; persistent right pleural effusion/thickening similar to previous examination; left lung remains clear.
DX 58 (EX 9)	11/9/99	Illuri	Cardiomegaly without congestive heart failure; pulmonary fibrosis; COPD.
DX 58 (EX 9)	4/6/00	Chiaradonna	COPD and chronic interstitial fibrotic changes as noted; somewhat progressive left pleural effusion increased in interim since previous study as well as subsegmental atelectatic changes about superior lingular segment; rule out elements of superimposed interstitial pneumonitis at right base; elements of redistribution of vascularity to apices are seen and compatible with elements of mild cardiac decompensation.
DX 58 (EX 9)	9/12/00	Chichakly	Worsening in appearance of lungs and heart since 4/6/00; rule out impending cardiac decompensation.
DX 58 (EX 9)	10/27/00	Chichakly	Residual changes, especially at lingula following CABG surgery and installation of mitral valve

Exhibit	Date of X-ray	Physician	Interpretation
			prosthesis; mild cardiomegaly, but congestive changes present on previous exam no longer seen; as expected, emphysema and fibrosis noted at this age.
DX 58 (EX 9)	1/12/01	Cohen	Interval worsening of congestive heart failure with bilateral pleural effusion, underlying infiltrates cannot be excluded; COPD changes, unchanged from prior exam.
DX 58 (EX 8)	1/19/01	Capati	Development of congestive heart failure and mild bilateral pleural effusions; moderate diffuse cardiac enlargement; COPD.
DX 58 (EX 9)	2/26/01	Cohen	Resolution of congestive heart failure with resolution of right chest wall loculated pleural effusion; blunting of CP angles bilaterally which might be related to small residua of pleural effusion versus pleural scars; COPD changes with bilateral fibrotic and scarring lung parenchyma, more on left side.
DX 58 (EX 9)	6/22/01	Cohen	COPD changes with old calcified granulomatous changes; interval increase in interstitial lung pattern which can be related to congestion or underlying pneumonitis; question of small pleural effusions versus pleural scarring.
DX 58 (EX 9)	7/5/01	Pine	Status post cardiac surgery, unchanged; extreme COPD with resolution of prior lingular bronchopneumonia.
DX 58 (EX 5)	3/1/02	Gatla	COPD; small pleural effusions versus pleural thickening; focal atelectasis left mid chest; cardiomegaly.
DX 58 (EX 5)	4/18/02	Gulati	Status post cardiac valve surgery; cardiomegaly; right lower lung consolidation; bilateral pleural effusions; COPD; focal atelectasis left mid chest; no mediastinal lesions.
DX 58 (EX 5)	6/7/02	Gulati	Sternotomy wire sutures are seen; cardiomegaly noted; right lower lung consolidation and pleural effusion; hyperaeration of left lung; COPD; focal atelectasis left mid lung.

CT Scan Evidence:

An October 6, 1999 CT scan of the chest with and without contrast by Dr. Theodore Gleason noted impressions of extensive abnormal lung parenchymal consolidation involving both lower lobes associated with small to moderate left pleural effusion and tiny right effusion. DX 58 (EX 6). Also noted was an approximately 3.5 by 2 CM nodular opacity abutting the paravertebral pleura in the posteriomedial right lung base which possibly represented malignant neoplasm and a linear opacity in the left upper lobe felt to likely represent chronic atelectasis and/or scarring.

A November 24, 1999 CT scan of the chest by Dr. W. Meeks noted impressions of bilateral emphysematous scarring and emphysematous blebs of lungs and left pleural thickening. DX 34A.

An April 27, 2000 CT scan of the chest without contrast by Dr. David W. Bean noted impressions of diffuse emphysematous changes both lungs, complete clearing of previously noted parenchymal process seen in posterior lungs bilaterally, and scarring within both lungs which was unchanged from previous exam. DX 58 (EX 6)

A January 10, 2002 CT scan of the chest without contrast by Dr. Crabtree noted impressions of moderate to severe diffuse emphysematous change, bilateral pleural fluid which is extended into the major fissures, improvement in infiltrates in both lung bases since previous study, and fibrotic change in both lung bases. DX 58 (EX 7)

H. Death Certificate

According to the Miner's death certificate, Thomas M. Burns died on June 7, 2002 in Carbondale, Jackson County, Illinois at the Memorial Hospital of Carbondale. DX 38. He was noted as being married and was survived by Mildred E. Kuehn (Burns). *Ibid*. The immediate cause of death was listed as right lower lobe pneumonia, with pneumoconiosis noted as a condition which gave rise to the immediate cause of death. Other significant conditions contributing to death but not resulting in the underlying cause of death were noted as diabetes, atrial fibrillation, and heart disease. *Ibid*. The death certificate was signed by Dr. Ana O. Migone.

Mr. Burns' Subsequent Claim

As noted above, a subsequent claim for benefits "shall be denied unless the claimant demonstrates that one of the applicable conditions of entitlement . . . has changed since the date upon which the order denying the prior claim became final." 20 C.F.R. § 725.309(d); *Peabody Coal Co. v. Spese*, 117 F.3d 1001 (7th Cir. 1997); *see also Allen v. Mead Corp.*, 22 B.L.R. 1-61 (2000) (claimant must establish by preponderance of evidence developed subsequent to denial of prior claim at least one element previously adjudicated against him). If the claimant demonstrates a change in one of the applicable conditions of entitlement, the findings made in connection with the prior claim are not binding in the adjudication of the subsequent claim. 20 C.F.R. § 725.309(d)(4).

The Miner's prior claim was denied in 1987 based on a finding that he had failed to meet any element of entitlement. DX 32. The evidence submitted since then clearly establishes a change in the Miner's condition. Indeed, Employer has now conceded that prior to his death the Miner suffered from pneumoconiosis arising out of his coal mine employment and contests only the issue of whether the Miner was, while still alive, totally disabled due to pneumoconiosis. Employer's Post-Hearing Memorandum ("Emp. Br.") at 40. I thus find that the Miner has established a change in one or more of the applicable conditions of entitlement and the claim is reopened.

Total Disability due to Pneumoconiosis

The regulations applicable to the Miner's claim, state, with respect to the issue of total disability:

> (b) *Total disability defined*. A miner shall be considered totally disabled if the irrebuttable presumption in § 718.304 applies. If the irrebuttable presumption described in § 718.304 does not apply, a miner shall be considered totally disabled if pneumoconiosis as defined in § 718.201 prevents or prevented the miner:
>
> (1) From performing his or her usual coal mine work; and
>
> (2) From engaging in gainful employment in the immediate area of his or her residence requiring the skills or abilities comparable to those of any employment in a mine or mines in which he or she previously engaged with some regularity over a substantial period of time.
>
> (c) *Criteria*. In the absence of contrary probative evidence, evidence which meets the standards of either paragraphs (c)(1), (2), (3), (4) or (5) of this section shall establish a miner's total disability:

20 C.F.R. § 718.204(b)-(c).[7]

A. Complicated Pneumoconiosis.[8]

Under 20 C.F.R. § 718.304, there is an irrebuttable presumption that a miner is totally disabled due to pneumoconiosis if the miner is suffering from complicated pneumoconiosis. Complicated pneumoconiosis is established by x-rays classified as Category A, B, or C, or by an autopsy or biopsy, which yields evidence of massive lesions in the lung or nodules in the lung that would equate to a one centimeter or greater opacity on x-ray. The determination of whether the miner has complicated pneumoconiosis is a finding of fact, and the administrative law judge must consider and weigh all relevant evidence. *Melnick v. Consolidation Coal Co.,* 16 B.L.R. 1-31 (1991); *Maypray v. Island Creek Coal Co.*, 7 B.L.R. 1-683 (1985).

[7] The regulations also provide that a miner who was employed for fifteen years or more in one or more underground coal mines, and who has a totally disabling respiratory or pulmonary impairment, shall be entitled to a rebuttable presumption that he is totally disabled due to pneumoconiosis. 20 C.F.R. § 718.305. The record in this case shows that the Miner worked in underground mines for only 13 years and in above-ground mining for an additional 27 years. DX 2, 3. Claimant has not met her burden of demonstrating that the surface mine dust conditions of the Miner's employment were substantially similar to those found in underground mines, and she is thus not entitled to the referenced presumption. *See Blakley v. Amax Coal Co*., 54 F.3d 1313 (6th Cir. 1995).

[8] Although Claimant's counsel asserts in her brief that Employer's stipulation to the presence of pneumoconiosis entitles Claimant to the irrebuttable presumption of § 718.304, Claimant's Post-Hearing Brief ("Cl. Br.") at 9-10, I do not view Employer's stipulation so broadly. At the hearing, Employer's counsel expressly contested the issue of total disability due to pneumoconiosis in the Miner's claim which necessarily means that he was not conceding the presence of complicated coal workers' pneumoconiosis since such a concession would be inconsistent with the sole issue upon which he is proceeding.

(i) X-ray Evidence of Complicated Pneumoconiosis.

The record contains six chest x-rays dated between November 1986 and October 2000. Viewing this evidence in its entirety, these six chest x-rays do not support a finding of complicated pneumoconiosis.

The chest x-ray dated November 4, 1986 was interpreted by five physicians, none of whom read it as showing complicated pneumoconiosis. DX 28, DX 32, DX 34C, EX 11.

A November 9, 1999 chest x-ray was determined by two physicians to be unreadable. EX 19, CX 1. This x-ray therefore does not support a finding of complicated pneumoconiosis.

A November 22, 1999 chest x-ray was interpreted by six physicians, two of whom read it as showing complicated pneumoconiosis. Dr. Ahmed, a dually-qualified physician interpreted the x-ray as 2/1, Category A while Dr. Cappiello, another dually-qualified physician read it as 3/2, Category A. DX 34A. Drs. Gaziana and Westerfield, both B-readers, read the November 22, 1999 x-ray as showing simple pneumoconiosis, as did Dr. Forry, a dually-qualified physician. DX 8, DX 28, DX 34C. Dr. Wiot, another dually-qualified physician, read this x-ray as completely negative for pneumoconiosis. EX 11. Considering the balanced qualifications of the reviewing physicians, and given the greater number of negative interpretations, I find the November 22, 1999 x-ray does not support a finding of complicated pneumoconiosis.

An April 6, 2000 chest x-ray of the Miner was found to be unreadable by Drs. Cohen and Wiot. CX 2, EX 19. This x-ray therefore does not support a finding of complicated pneumoconiosis.

A September 12, 2000 chest x-ray was read by Dr. Cohen, a B-reader, as revealing simple pneumoconiosis. CX 3. The same x-ray was found to be unreadable by Dr. Wiot, a dually-qualified physician. EX 19. Neither interpretation supports a finding of complicated pneumoconiosis.

The most recent chest x-ray of the Miner was taken October 13, 2000, and interpreted by six physicians, only one of whom found it to be positive for complicated pneumoconiosis. Dr. Miller, a dually-qualified physician, read the x-ray as revealing 2/2, Category A pneumoconiosis. Dr. Cappiello, another dually-qualified physician, read the x-ray as "2/2"[9] after having interpreted the earlier November 22, 1999 x-ray as showing Category A complicated pneumoconiosis. DX 34B. The remaining four physicians, two of whom are dually-qualified, read the October 13, 2000 chest x-ray either as completely negative for the disease or showing only simple pneumoconiosis. DX 26, DX 34B, CX 4, EX 19. Considering the qualifications of the reviewing physicians, the number of interpretations which were negative for complicated pneumoconiosis, and the fact that Dr. Capiello, Claimant's own dually-qualified expert, found this x-ray did not show evidence of complicated pneumoconiosis despite having interpreted the

[9] Category 2 is an indication of numerous small opacities but normal lung markings still visible. References to Category A through C opacities denote the presence of complicated pneumoconiosis, *i.e.*, large opacities of one centimeter in diameter or greater.

earlier November 22, 1999 x-ray as showing complicated pneumoconiosis, I find that the October 13, 2000 x-ray does not support a finding of complicated pneumoconiosis.

Neither the CT scan evidence nor the chest x-ray evidence contained in the Miner's treatment and hospitalization records summarized above support a finding of complicated coal workers' pneumoconiosis.

Based on the foregoing, I find the chest x-ray evidence is insufficient to invoke the irrebuttable presumption of § 725.304(a).

(ii) Biopsy/Autopsy Evidence of Complicated Pneumoconiosis.

The October 6, 1999 bronchoscopy and biopsy performed by Dr. Crabtree revealed no evidence of complicated coal workers' pneumoconiosis. EX 20. Similarly, the autopsy performed by Dr. O'Neill on June 7, 2002, resulted in a diagnosis of coal workers' pneumoconiosis but no reference to any evidence of massive lesions or nodules in the lung that would equate to a one centimeter or greater opacity on x-ray. DX 41. Indeed, Dr. O'Neill's report contains no measurements of any nodules or lesions he observed.

With respect to the physicians who reviewed the biopsy and autopsy slides, Dr. Naeye rendered a diagnosis of simple, not complicated, coal workers' pneumoconiosis. DX 58 (EX 4) at 3. Dr. Caffrey similarly found a moderate degree of simple, but not complicated, coal workers' pneumoconiosis. EX 13 at 6. Dr. Oesterling also found evidence of only "mild micronodular coal workers' pneumoconiosis." EX 14 at 6-7. Dr. Crouch likewise concluded that the Miner suffered from only simple coal workers' pneumoconiosis. EX 21B.

Dr. Perper is the only physician who, based on his review of the autopsy slides, noted "[m]ultiple pneumoconiotic micronodules and macronodules measuring up to 1.1 cm." DX 68 at 27. However, despite his reference to macronodules measuring up to 1.1, Dr. Perper diagnosed "[c]oal workers' pneumoconiosis, *simple*, moderate to severe, with micro-nodules and macronodules, mixed coal dust and silicotic types, and birefringent silica crystals." *Ibid*. (italics added). Indeed, Dr. Perper repeated, in the conclusion to his report:

> [A]fter reviewing the above documentation and materials it is my professional opinion within a reasonable degree of medical certainty that:
>
> > 1. Mr. Burns had evidence of significant and substantial, severe *simple* coal workers' pneumoconiosis, causally associated with centrilobular emphysema.
>
> . . . . .

Id. at 36 (italics added).

A review of Dr. Perper's forty-four page report makes clear that he is aware of the distinction between simple and complicated coal workers' pneumoconiosis, and his use of the term "simple" in connection with the Miner's coal workers' pneumoconiosis is thus presumed to

be intentional. Furthermore, even if I were to assume that his findings of "macronodules measuring up to 1.1 cm" during microscopic examination of the autopsy slides are evidence of "massive lesions" in the Miner's lungs, Dr. Perper did not offer any opinion on whether these macronodules "would equate to a one centimeter or greater opacity on x-ray." 20 C.F.R. § 718.304. I thus have no basis upon which to determine whether these macronodules would constitute a 1.0 centimeter or greater opacity on a chest x-ray. *See, e.g.*, *Double B Mining, Inc. v. Blankenship*, 177 F.3d 240 (4th Cir. 1999)(finding of "massive fibrosis" on biopsy, which included a lesion measuring 1.3 centimeters in diameter, insufficient to determine whether miner had complicated pneumoconiosis); *Smith v. Island Creek Coal Co.*, 7 B.L.R. 1-734 (1985)(no attempt made to equate nodules found on autopsy with size of x-ray opacities). I therefore find that Dr. Perper's opinion is insufficient to establish the presence of complicated pneumoconiosis.

Based on all the evidence, I thus find that the Miner is not entitled to the irrebuttable presumption of § 718.304.

B. Inability to Perform Coal Mine or Similar Work.

As noted above, even though complicated coal workers' pneumoconiosis has not been established, benefits may still be awarded if the evidence shows the Miner was unable to perform his prior coal mine employment prior to death because of a totally disabling respiratory or pulmonary condition caused or contributed to by his exposure to coal dust. Evidence relevant to this issue includes pulmonary function tests, arterial blood-gas tests, evidence of cor pulmonale with right sided congestive heart failure, and physicians' reasoned medical judgment. 20 C.F.R. § 718.204(c)(1)-(4).

With respect to the pulmonary function tests of record, the earliest test of record is the study performed November 4, 1986 which does not establish disability. DX 32. Of the remaining fifteen studies, most or all of the physicians who reviewed them, as Claimant acknowledges, Cl. Br. at 21, found eight of them to be invalid.[10] Of the seven valid pulmonary function studies, those performed on November 9, 1999, April 6, 2000, September 12, 2000, October 13, 2000, and October 26, 2000 revealed one or more qualifying values supportive of total disability.[11] The studies performed on November 22, 1999 and April 27, 2000 did not produce qualifying results. The pulmonary function results overall thus support a finding of total disability.

With respect to the arterial blood-gas tests, the results from tests performed on October 6, 1999, October 22, 1999, and June 7, 2002 support a finding of total disability. DX 34A, DX 58 (EX 5), DX 58 (EX 7). The remaining six studies did not produce qualifying results. Overall, therefore, the arterial blood-gas results do not support a finding of total disability.

[10] The eight invalid pulmonary function tests are: July 16, 1997 (DX 58); October 6, 1999 (DX 34A); January 11, 2001 (DX 34A); February 22, 2001 (DX 34A); April 6, 2001 (DX 34A); June 21, 2001 (DX 34A); July 5, 2001 (DX 58), and August 2, 2001. Cl. Br. at 21.

[11] The pulmonary function study tables found in Part 718, Appendix B of the regulations reflect values for individuals up to age 71. Since the Miner died at age 79, it was therefore necessary to obtain supplemental information from the Office of Workers' Compensation Programs, Division of Coal Mine Workers' Compensation, for individuals age 72 through 79. Copies of this information were provided to counsel for the parties prior to the issuance of this decision.

With respect to the medical opinion evidence, only one physician, Dr. Douglas Thomson, concluded that the miner was not disabled. DX 32. He reached that conclusion after he examined the Miner on November 5, 1986 at the time of his original claim for benefits. All physicians who either examined the Miner or reviewed the medical evidence in connection with the Miner's subsequent claim have concluded that he is totally disabled. DX 6, 17, 19, 26, 34C, 42, 58, 68 ; CX 5, 8; EX 4, 12, 13, 14, 17, 18, 21. There is a clear divergence of opinions, however, with respect to the cause of the Miner's disability. Four physicians, Drs. Sanjabi, Crabtree, Perper, and Cohen, attribute the Miner's pulmonary disability in whole or in part to his coal dust exposure. DX 6, 17, 19, 42, 68; CX 5, 8. In contrast, eight physicians, Drs. Tuteur, Westerfield, Naeye, Caffrey, Oesterling, Repsher, Crouch, and Fino, have concluded that the Miner was totally disabled due to one or more conditions which had nothing to do with his coal mine employment. DX 26, 34C, 58; EX 4, 12, 13, 14, 17, 18, 21A, 21 B. For the reasons set forth below, I find that the opinions of Drs. Perper and Cohen are the most persuasive and that they support a finding of total disability caused in whole or substantial part by his coal mining employment.

Dr. Perper reviewed the available medical evidence of record, performed a pathological examination of the Miner's biopsy slides, and opined in his August 20, 2003 report that the Miner's respiratory condition is attributable to coal dust exposure. DX 68. He concluded that the Miner had "substantial and significant coal workers' pneumoconiosis" based on: his 40 years of coal mining; clinical symptoms; pulmonary function and blood gas test results showing restrictive and obstructive defects, abnormal pulmonary diffusion, and hypoxemia; chest x-ray evidence; clinical diagnoses of COPD and coal workers' pneumoconiosis by other physicians; and severe and significant coal workers' pneumoconiosis shown at autopsy. *Id.* at 30-31. He further concluded that the Miner's respiratory impairment was totally disabling and that his coal workers' pneumoconiosis and associated centrilobular emphysema were substantial contributing causes of disability. *Id.* at 33-35. With respect to the Miner's centrilobular emphysema, Dr. Perper recognized that, while smoking is a known complication of the disease, the Miner quit smoking in 1958, forty-four years prior to his death. *Id.* at 31. He also noted that "the manifestations of COPD and emphysema became apparent only in 1986, twenty-eight years after the miner quit smoking . . . [and it] is well known that there is no further damage and progression of lung damage due to smoking, after an individual stop[s] smoking." *Ibid*. He further wrote that "it is well known and documented in the medical literature that coal workers' pneumoconiosis and associated pulmonary damage may progress years after cessation of occupational exposure to coal dust-containing silica." *Ibid*. Dr. Perper's report is both well documented and well reasoned, and I thus find it is entitled to substantial weight.

Dr. Cohen, like Dr. Perper, concluded that the Miner suffered from a disabling respiratory impairment attributable to his coal dust exposure. Dr. Cohen determined that the Miner had a restrictive and severe obstructive lung defect, as well as diffusion impairment and gas exchange abnormalities prior to his death. CX 5 at 23. He further concluded that the Miner's 40 year exposure to coal dust was a primary cause of his pulmonary disability, and that his limited exposure to tobacco smoke was too insignificant and remote to be a contributing factor. *Ibid*. It was also his opinion that the Miner, because of his respiratory condition, could not have tolerated the dusty atmosphere of a coal mine nor could he perform the physical duties

of his last coal mining job. *Ibid*. With respect to his conclusion that the Miner's obstructive lung disease was due to coal dust exposure, he noted numerous studies, accepted as authoritative by, *inter alia*, the Department of Labor and NIOSH, that show a relationship between the development of COPD, manifested as chronic bronchitis and emphysema, and coal dust exposure. *Id.* at 23-26. According to Dr. Cohen:

> There are no similar types of scientific study refuting any of the conclusions reached by these well-known and respected scientists and researchers. The old notion that coal dust produces only the classic restrictive pattern of lung disease is simply no longer acceptable in light of this abundant epidemiological research. Clearly, coal dust causes obstructive lung disease, like it has done to Mr. Burns.

Id. at 25. Dr. Cohen's opinion, like that of Dr. Perper, is both well documented and well reasoned, and I give it substantial weight.

In contrast to the opinions of Drs. Cohen and Perper, eight physicians (Drs. Tuteur, Westerfield, Crouch, Naeye, Caffrey, Oesterling, Repsher, and Fino) have concluded based on their review of the available medical evidence and/or their examination of the Miner, that his disability was neither caused nor contributed to by exposure to coal dust. Each are discussed below.

Opinion of Dr. Tuteur

Dr. Tuteur examined the Miner on October 13, 2000 and concluded that the miner suffered from "extensive intrathoracic disease . . . [which was] predominantly post inflammatory pleural and parenchymal disease in association with motor vehicle accident, recurrent pneumonias, and coronary artery bypass grafts, associated with aortic valve replacement." DX 26 at 3. He noted that pulmonary function testing revealed a severe obstructive ventilatory defect and abnormal gas exchange during exercise, and that the Miner was totally and permanently disabled, but concluded that his disability was "in no way related to, aggravated by, or caused by the inhalation of coal mine dust or the development of coal workers' pneumoconiosis." *Ibid*.

On March 7, 2005, Dr. Tuteur conducted a review of the available medical evidence and concluded that, at the time of his death, the Miner had simple coal workers' pneumoconiosis. EX 21A at 13. He further opined that the Miner's coal workers' pneumoconiosis "was of insufficient severity and profusion to produce clinical symptoms, physical examination abnormalities, impairment of pulmonary function, or even significant radiographic change." *Ibid*. He attributed the Miner's significant pulmonary impairment in large part to complications including necrotizing pneumonia following treatment for coronary artery disease, including the Miner's coronary artery bypass graft in 1997. *Ibid*. He also took issue with Dr. Perper's conclusion that the Miner had a combined obstructive and restrictive ventilatory defect, stating that an absolute criteria for the diagnosis of a restrictive abnormality is a reduced total lung capacity and all measurements of the Miner's total lung capacity in this case were normal. *Ibid*. Furthermore, he stated that Dr. Oesterling improperly equated centrilobular emphysema with focal emphysema, and "[c]entrilobular emphysema tends to be a destructive phenomenon either

due to necrotizing pneumonias and/or chronic inhalation of tobacco smoke." *Id.* at 14. Dr. Tuteur also noted that x-ray interpretations from 1986 and 1987 reported a profusion of only 0/0 or 1/1 opacities, 1986 pulmonary function studies revealed no disabling impairment of pulmonary function, and subsequent impairment was due to a combination of thoracic trauma associated with an automobile accident and complications of surgical treatment following coronary artery bypass graft and aortic valve replacement. *Ibid*.

According to Dr. Perper, Dr. Tuteur's opinion is flawed, *inter alia*, because autopsy findings clearly showed significant and substantial coal workers' pneumoconiosis. DX 68 at 12. In a supplemental report dated March 23, 2005, Dr. Perper noted that the autopsy prosector, Dr. O'Neill, recorded, *inter alia*, marked anthracotic deposits in both lungs and diagnosed coal workers' pneumoconiosis and findings consistent with a clinical history of severe chronic obstructive pulmonary disease. CX 8 at 16. He further noted that Dr. Naeye, like Dr. Perper, concluded that the Miner suffered from both restrictive and obstructive lung disease, and that Dr. Tuteur improperly ignored the causal connection between the Miner's severe emphysema and COPD and his coal workers' pneumoconiosis. *Ibid*.

Similarly, Dr. Cohen criticized Dr. Tuteur's opinion because: (1) Dr. Tuteur did not review any medical records relating to the Miner's 1989 motor vehicle accident and his conclusion linking any of the Miner's respiratory impairment to that incident was thus speculative; (2) Dr. Tuteur noted in his report, but ignored in his conclusion, the fact that the Miner suffered from shortness of breath as early as 1985, four years before the accident, which could not be explained by the trauma suffered in 1989; and (3) any lung damage caused by the trauma from an automobile accident would appear *restrictive* in nature and the Miner's impairment was primarily obstructive in nature. CX 5 at 27-28. Dr. Cohen further noted that Dr. Tuteur's attribution of any of the Miner's respiratory condition to his cardiac surgery was similarly flawed since the records of Dr. Crabtree, the Miner's treating pulmonary specialist, and Dr. Moses, his treating cardiologist, revealed that the Miner recovered completely from the surgery and did not have any ongoing problems related to the procedure. *Id.* at 28.

For a variety of reasons, including the following, I am not persuaded by Dr. Tuteur's opinion. First, while Dr. Tuteur attributes the Miner's significant pulmonary impairment to complications following surgery for his cardiac problems and, to a lesser degree, the trauma sustained in the 1989 automobile accident, the records of the Miner's own treating cardiologist and pulmonologist, as Dr. Cohen notes, refute Dr. Tuteur's conclusion. For example, Dr. Moses wrote in a letter dated October 8, 1999 that the results of a bronchoscopy performed by Dr. Crabtree on October 6, 1999 for hemoptysis revealed "the problem appears to be mainly one of bronchiectasis and coal miner's lung." (DX 58 – EX 6). Similarly, in a letter dated April 27, 2000, Dr. Crabtree noted that the Miner suffered from significant bronchiectasis and bronchial obstructive lung disease consistent with his previous exposure to coal dust and his condition would likely decline in the future. DX 19. Second, as Dr. Cohen also noted, any pulmonary impairment resulting from the automobile accident would have manifested itself as a *restrictive* disease, and, according to Dr. Tuteur, the medical evidence of record does not establish that the Miner had *any* restrictive disease. This conclusion of Dr. Tuteur is itself inconsistent with several other physicians' opinions including those of Dr. Westerfield (DX 34C – "moderate to severe restrictive lung disease"); Dr. Naeye (DX 58 – "1997 cardiac aortic valve surgery led to

scarring of lung parenchyma with resultant restrictive lung disease in a localized area of one lung"); and Dr. Crabtree (DX 58 – 10/6/99 pulmonary function test "suggests combined obstructive and restrictive defect with severe obstruction and moderate restriction with severe reduction in diffusing capacity consistent with diagnosis of CWP"). Finally, Dr. Tuteur's statement that "[c]entrilobular emphysema tends to be a destructive phenomenon either due to necrotizing pneumonias and/or chronic inhalation of tobacco smoke" (EX 21A at 14) suggests that Dr. Tuteur does not accept the proposition that centrilobular emphysema may be associated with exposure to coal dust. Such a conclusion is inconsistent with the prevailing view in the medical community upon which the Department of Labor relied in formulating the regulations governing Black Lung disability claims which became effective on January 19, 2001. Although those regulations do not govern this claim, the medical literature cited and relied upon by the Department clearly predates the revised regulations and was in existence at the time the Miner filed this claim on October 22, 1999. For example, in its comments regarding the adoption of the revised regulations, the Department notes, in relevant part:

> Drs. Fino and Bahl find no scientific support that clinically significant emphysema exists in coal miners without progressive massive fibrosis, . . . but the available pathologic evidence is to the contrary. Cockcroft evaluated 39 coal workers and 48 non-coal worker controls dying of cardiac causes in 1979. . . . Centrilobular emphysema (the predominant type observed) was significantly more common among the coal workers. The severity of the emphysema was related to the amount of dust in the lungs. These findings held even after controlling for age and smoking habits.

Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969, as Amended, 65 Fed. Reg. 79920, 79941 (Dec. 20, 2000) ("Final Rule") (internal citations omitted).[12] The Department went on to write:

> Similarly, Leigh and colleagues analyzed 886 miners who died between 1949 and 1982. . . . . They found that miners with more years of face work had worse emphysema pathologically. In a subsequent study of 264 underground coal miners exposed to mixed coal and silica dust, Leigh . . . made the following important findings: (1) The extent of emphysema was strongly related to the total coal content of the lung, age and smoking; (2) in miners who were lifelong non-smokers, the extent of emphysema was strongly related to coal content and age; (3) the extent of emphysema was unrelated to lung silica content; and (4) the extent of lung fibrosis was related to silica content. The authors concluded that

[12] It is not unusual for courts to cite to, and consider, published comments underlying the promulgation of regulations. *See Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 156 n. 29 (1988) (favorable discussion of Department's comments underlying promulgation of 20 C.F.R. § 727.203(a) to determine that agency did not intend that a single piece of qualifying evidence would be sufficient to invoke interim presumption); *Consolidation Coal Co. v. Director, OWCP [Stein]*, 294 F.3d 885, 892 (7th Cir. 2002) (favorable consideration of Department's December 2000 comments with regard to use of CT-scans in assessing presence or absence of pneumoconiosis); *Bonessa v. United States Steel Corp.*, 884 F.2d 726, 729 (3rd Cir. 1989) (favorable referral to Department's 1983 comments to 20 C.F.R. § 718.205(c) in assessing causation). Therefore, consideration of the Department's findings as set forth in the comments to the amended regulations is proper here.

> "these results provide strong evidence that emphysema in coalworkers is causally related to lung coal content."
>
> Ruckley and colleagues achieved similar results in examining the lungs of 450 coal workers to determine the association between coal mine dust exposure and dust-related fibrosis and emphysema. . . . . The authors found emphysematous changes in 72% of miners who smoked, 65% of ex-smokers, and 42% of nonsmoking miners; emphysema scores were higher in patients with increasing evidence of pneumoconiotic disease; and increasing coal lung dust was associated with the presence of emphysema. Forty-seven percent of miners with no fibrotic lesions had emphysema. Ruckley concluded that "the results support the conclusion that the relationship observed between respirable dust and emphysema in coal workers is, in some way, causal."

Id. at 79941-42 (internal citations omitted). The Department also quotes several studies which establish a link between emphysema and bronchitis to coal dust exposure including the following:

> Most evidence to date indicates that exposure to coal mine dust can cause chronic airflow limitation in life and emphysema at autopsy, and this may occur independently of CWP * * * The relationships between hypersecretion of mucus (chronic bronchitis) and chronic airflow limitation (emphysema) on the one hand and environmental factor of coal mining exposure on the other appear to be similar to those found for cigarette smoking.

Id. at 79939

Both Drs. Perper and Cohen expressly relied on the medical literature cited by the Department of Labor in reaching their conclusion that the Miner's pulmonary impairment was linked to his 40 years of exposure to coal dust. *See, e.g.,* DX 68 at 30-44; CX 5 at 23-27. Based on the foregoing, I find the opinions of Drs. Perper and Cohen more persuasive, and thus entitled to more weight, than the contrary opinion of Dr. Tuteur.

Opinion of Dr. Westerfield

Dr. Westerfield reviewed the available medical evidence at Employer's request and opined in a letter dated September 26, 2001 that the Miner had simple coal workers' pneumoconiosis based on chest x-rays revealing category 1/1 opacities and "moderate to severe Restrictive lung disease." DX 34C at 6 (emphasis in original). It was Dr. Westerfield's further opinion, like that of Dr. Tuteur, that the Miner's pulmonary impairment was due primarily to complications of heart valve replacement surgery and the 1989 motor vehicle accident. *Ibid*.

As noted by Dr. Perper, Dr. Westerfield omitted from his opinion, and thus appears not to have considered, several significant facts including: the potential impact on the Miner's pulmonary condition of his 40 years of exposure to coal dust; recent chest x-ray interpretations noting category 2/1 and 3/2 opacities consistent with pneumoconiosis in all six lung zones; and

substantial evidence showing both restrictive *and* obstructive lung defects. DX 68 at 14-15. Indeed, Dr. Westerfield, unlike virtually every other physician, did not conclude that the Miner suffered from emphysema or any other form of COPD. DX 34C at 6. Furthermore, as Dr. Cohen noted, Dr. Westerfield did not have the opportunity to review records related to the Miner's treatment after his 1989 motor vehicle accident and the view that his pulmonary problems are related to the accident therefore appears speculative. CX 5 at 27. Similarly, Dr. Cohen noted that the Miner's treating pulmonologist and cardiologist (Drs. Crabtree and Moses) both concluded that the Miner completely recovered from his 1997 CAGB and heart valve replacement surgery, the only other cause cited by Dr. Westerfield for the Miner's pulmonary condition. CX 5 at 28. For these reasons, as well as those detailed above regarding Dr. Tuteur's opinion, I find Dr. Westerfield's opinion less persuasive than the opinions of Drs. Perper and Cohen and thus accord it less weight.

Opinion of Dr. Crouch

In a two-page letter dated February 12, 2003, Dr. Crouch noted that she had reviewed the Miner's autopsy slides and concluded that lung tissue revealed "coal dust deposition with histologic changes of simple coal workers' pneumoconiosis characterized by scattered coal dust macules and a micronodule." EX 21B at 2. She further concluded that, "[g]iven the mixed patterns of emphysema, the major risk factor for alveolar destruction was cigarette smoking." *Ibid*. Dr. Crouch also opined that "[t]he etiology of the [Miner's] pulmonary vascular changes is uncertain but most likely reflects some combination of this patient's cardiac and obstructive lung disease." *Ibid*.

Like Dr. Tuteur, Dr. Crouch cites only cigarette smoking as "the major risk factor" for the Miner's emphysema. Similarly, in contrast to the opinions of the Miner's treating pulmonologist and cardiologist, she attributes the Miner's decline to his heart-related surgery and automobile accident and opines that "coal dust deposition could not have caused a clinically significant degree of functional impairment or disability . . . ." EX 21B at 2. For all the reasons set forth above, I accord less weight to her opinion than the opinions of Drs. Perper and Cohen.

Opinion of Dr. Naeye

In a report dated April 20, 2003, Dr. Naeye noted that he had reviewed extensive medical evidence relating to the Miner, including tissue slides, and concluded that, although Mr. Burns suffered from coal workers' pneumoconiosis, the size and extent of the associated lesions in the lung tissue were "far too small for them to have had any measurable effect on lung function . . . ." EX 4 at 2. According to Dr. Naeye, the lung tissues removed at autopsy had anthracotic lesions with associated black pigment which, upon close inspection, "strongly suggests that the association is not the result of toxic products in coal mine dust." *Id.* at 3. Furthermore, Dr. Naeye concluded that Mr. Burns' pulmonary function results were "characteristic of many ex-coal miners who smoked cigarettes in their earlier life, developed chronic bronchitis as well as slowly progressive centrilobular emphysema which eventually led to disability and sometimes death." *Ibid*. He thus concluded that "cigarette smoking had the dominating role in causing his chronic bronchitis, progressive centrilobular emphysema and death." *Ibid*.

Both Dr. Perper and Dr. Cohen took issue with Dr. Perper's opinion. For example, Dr. Perper noted that Dr. Naeye took note of Mr. Burns' history of shortness of breath but omitted from the clinical findings in his report significant information regarding the Miner's other respiratory symptoms such as evidence of cough, expectoration of mucus, and hemoptysis. DX 68 at 21. He further noted that Dr. Perper mentioned only chest x-rays in the last years of the Miner's life which were interpreted as showing diffuse bilateral emphysema when, in fact, there were earlier markedly abnormal chest x-rays in the medical evidence. *Ibid*. With respect to Dr. Naeye's attribution of the Miner's chronic bronchitis and progressive centrilobular emphysema to cigarette smoking, Dr. Perper noted that "[i]t is well and universally agreed and scientifically documented that upon cessation of cigarette smoking there is no further lung damage, because exposure to noxious cigarette[] fumes ceases." *Id.* at 25. Dr. Perper goes on to note that Mr. Burns stopped smoking in 1953 and a pulmonary function study done some 33 years[13] later on November 4, 1986 was "totally normal." *Ibid*. He wrote:

> It is totally unreasonable and unacceptable to suggest that after [33] years of non-smoking with documented normal pulmonary function tests, smoking suddenly starts to play "a dominant role" in causing the patient's pulmonary disability and death.

Ibid. Dr. Perper further noted that, in contrast to the diminishing effects of smoking after an individual stops smoking, "it is well known and documented in the medical literature that coal workers' pneumoconiosis and associated pulmonary damage may progress years after cessation of occupational exposure to coal dust-containing silica." *Id.* at 31.[14] Dr. Cohen, like Dr. Perper, criticizes Dr. Naeye's attribution of the Miner's pulmonary condition to smoking and relies on a multitude of medical studies which link both chronic bronchitis and emphysema to exposure to coal dust. CX 5 at 23-26. According to Dr. Cohen:

---

[13] Dr. Perper erroneously states that the November 4, 1986 pulmonary function study was done only 28 years after the Miner stopped smoking. DX 68 at 25. Since Mr. Burns stopped smoking in 1953, *see, e.g.*, Tr. 34, 41, there was actually a lapse of 33 years between the two events. However, this mathematical error on Dr. Perper's part would clearly not change his opinion in light of the fact that there was an even greater length of time between when Mr. Burns stopped smoking and developed a respiratory impairment.

[14] *See also* Dr. Perper's discussion of the relevant medical literature, including that relied upon and cited by the Department of Labor in the Final Rule published December 20, 2000 in the Federal Register, substantiating the fact that pneumoconiosis may develop long after an individual stops working in the mines. DX 68 at 32-33. For example, the Department cited a study where "[t]he authors found emphysematous changes in 72% of miners who smoked, 65% of ex-smokers, and 42% of nosmoking miners . . . ." Final Rule, 65 Fed. Reg. at 79942. The Department further noted in the comments accompanying its Final Rule:

> [T]here is . . . evidence that lung function can continue to deteriorate after a miner leaves the coal mining industry.
>
> . . . . .
>
> [One study] demonstrated a decline of pulmonary function in both smoking and non-smoking coal miners that continues over time even after retirement from mining. Given this evidence of progression, it is clear that a miner who may be asymptomatic and without significant impairment at retirement can develop a significant pulmonary impairment after a latent period.

Id. at 79971 (citations omitted).

> There are no similar types of scientific study refuting any of the conclusions reached by these well-known and respected scientists and researchers. The old notion that coal dust produces only the classic restrictive pattern of lung disease is simply no longer acceptable in light of this abundant epidemiological research. Clearly, coal dust causes obstructive lung disease, like it has done to Mr. Burns.

Id. at 25. Dr. Cohen further notes that many studies have shown that the respiratory symptoms reported by Mr. Burns, such as cough, sputum production, shortness of breath, and wheezing are related to either the duration of exposure or cumulative exposure to coal dust. *Id.* at 26. Like Dr. Perper, Dr. Cohen also concluded that: "The presence of severe impairment in Mr. Burns was definitely a result of his coal dust exposures and not his remote and limited smoking history."

On December 4, 2003, Dr. Naeye authored another report in which he responded to the various criticisms by Dr. Perper of his opinion. EX 12. He noted, in part, that "Dr. Perper is a forensic pathologist who has a good record of publications in forensic pathology and no record of any research or publications in the field of coal workers' pneumoconiosis (CWP)." *Id.* at 1. Dr. Naeye further noted that they had both reviewed the same clinical information, but he concluded that "Dr. Perper displays a lack of knowledge about the complex pathogenesis of CWP that has led him [to] misinterpret findings in the lungs of Thomas Burns." *Ibid*. Dr. Naeye agreed with Dr. Perper that Mr. Burns had clinically significant lung disease when he died, but noted that pulmonary function and arterial blood gas test results from 1985 when he retired from coal mining were normal and it was only later that he developed severe abnormalities in lung function. *Ibid*. Dr. Naeye further wrote that cigarette smoking "makes a *several-fold greater contribution to the genesis of centrilobular emphysema and chronic bronchitis than does prolonged exposure to coal mine dust*." *Ibid*. (emphasis in original). He also wrote: "*Once established by cigarette smoking centrilobular emphysema progresses, even when miners and ex-miners stop smoking.*" *Ibid*. (emphasis in original). Citing three studies published between 1973 and 1976, which conclude that "bronchitis has little or no effect on lung function unless the subject happens to be a cigarette smoker," and another study published in 1986 showing that "[a]irway obstruction caused by centrilobular emphysema and bronchitis that is severe enough to preclude a miner from working is very rare if indeed it occurs at all in the absence of smoking or complicated CWP," Dr. Naeye concluded that Mr. Burns' simple coal workers' pneumoconiosis did not cause disability. *Ibid*.

In a March 23, 2005 report, Dr. Perper responded at length to Dr. Naeye's December 4, 2003 supplemental opinion. CX 8. He noted, for example, that medical studies upon which Dr. Naeye relied were outdated and a large body of medical literature, including that relied on by the Department of Labor in adopting the revised regulations, recognizes that occupational exposure to coal mine dust results in centrilobular emphysema and COPD beyond any effect of smoking. *Id.* at 3. He further disputed Dr. Naeye's assertion that centrilobular emphysema progresses after quitting smoking, noting that "*after 20 years or so of smoking abstinence the risk of former smokers is very close to that of never smokers*." *Ibid*. (emphasis in original).

I note that, although Dr. Naeye states that centrilobular emphysema progresses "once established" even though a miner or ex-miner may stop smoking, he does not identify when this

disease process was "established" with respect to this Miner. According to his wife, Mr. Burns stopped smoking sometime in 1953 after having smoked about one pack of cigarettes a day for approximately eleven or twelve years.[15] For at least the next 33 years, there is no evidence of any lung impairment whatsoever. Clearly, the Miner's centrilobular emphysema was not "established" for a substantial period of time after Mr. Burns quit smoking, yet his exposure to coal dust continued for the next 32 years until he retired from coal mining in 1985. DX 37. Given the lapse of more than 30 years between smoking cessation and any evidence of pulmonary impairment, Dr. Naeye's failure to explain the basis for his conclusion that smoking played a "dominating role" in Mr. Burns' centrilobular emphysema substantially diminishes the value of his opinion, especially in light of the fact, noted by both Dr. Perper and Dr. Cohen, that damage to the lungs caused by smoking typically does not progress after smoking ceases. Furthermore, I note that Dr. Naeye substantially downplays the significance of an individual's exposure to coal mine dust as a causative factor in the development of emphysema. This view, as noted above, is contrary to the prevailing view in the medical community set forth in the studies cited in the Department's Final Rule. Based on the foregoing, I find the opinions of Drs. Perper and Cohen regarding the etiology of Mr. Burns' lung impairment to be better reasoned, and thus entitled to more weight, than the contrary opinion of Dr. Naeye.

Opinion of Dr. Caffrey

According to the written report of Dr. Caffrey dated December 29, 2003, he reviewed multiple medical records as well as autopsy and surgical slides relating to the Miner. EX 13. Based on his review of this evidence, he opined that Mr. Burns suffered from moderate simple coal workers' pneumoconiosis. *Id.* at 6. He further wrote:

> Definitely prior to his death Mr. Burns was totally disabled from his cardiac disease and his respiratory disease, but certainly he would not have been able to work anyway because he was 78 years of age. . . .

Id. at 7. He believed that Mr. Burns' pulmonary problems "were certainly accelerated following his motor vehicle accident in 1989 . . . ." *Ibid*. He also stated that some of the Miner's centrilobular emphysema "may be due to [his] simple CWP, but for the most part it does not appear that bronchitis or centrilobular emphysema were a debilitating problem no matter what the cause." *Id.* at 7-8. He believed that the Miner's severe cardiac problems and associated diabetes mellitus "could have caused" Mr. Burns' severe pulmonary problems, although he acknowledged "whether they alone did cause his severe pulmonary problems I certainly cannot objectively say." *Id.* at 8.

Dr. Perper responded to Dr. Caffrey's opinions in a March 23, 2005 supplemental report, finding Dr. Caffrey's report to be equivocal as to the role of coal workers' pneumoconiosis and emphysema in Mr. Burns' disability and death CX 8. Dr. Cohen similarly reviewed Dr. Caffrey's report and, although he did not comment directly on the opinions contained therein, he

[15] According to Mrs. Burns, her husband did not smoke before he joined the Navy after Pearl Harbor was attacked on December 7, 1941. Tr. 40. When he returned from the Navy in 1945, he was smoking. *Ibid*. He stopped smoking cigarettes around mid 1953. Tr. 34. Assuming that Mr. Burns started smoking shortly after he enlisted, he smoked cigarettes for approximately eleven or twelve years.

noted that he had not changed his opinion that Mr. Burns' 40 years of coal dust exposure was a significant contributing cause in the development of his severe and disabling lung disease. CX 7 at 2-3.

I find Dr. Caffrey's opinion to be both internally inconsistent and poorly documented. For example, after first opining that Mr. Burns was "[d]efinitely . . . totally disabled from his cardiac disease *and his respiratory disease*" before death, EX 13 a 7 (emphasis added), Dr. Caffrey contradicts himself by stating further in his report "it does not appear that bronchitis or centrilobular emphysema were a debilitating problem no matter what the cause." *Id.* at 7-8. Similarly, he notes initially that the Miner's centrilobular emphysema "may be due to [his] simple CWP," *id*. at 7, but then observes elsewhere that the Miner's severe cardiac problems and associated diabetes mellitus "could have caused" Mr. Burns' pulmonary problems. *Id.* at 8. Dr. Caffrey's use of phrases such as "could be," "could have," "I believe," and "it does not appear" also tend to diminish the value of his opinion. *See, e.g.*, *Griffith v. Director, OWCP*, 49 F.3d 184 (6th Cir. 1995) (treating physician's opinion entitled to little weight where he concluded miner "probably" had black lung disease); *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91 (1988) (equivocal opinion regarding etiology may be given less weight). Finally, I note that Dr. Caffrey does not cite any specific objective test results which he believes might support any of these contradictory conclusions. I thus find Dr. Caffrey's opinion, at least with respect to the issues of disability and causation, to be equivocal, poorly reasoned, contradicted by the better reasoned and documented opinions of Drs. Perper and Cohen, and entitled to little weight.

Opinion of Dr. Oesterling

In his February 4, 2004 report, Dr. Oesterling notes that, after reviewing autopsy and surgical slides, he determined, *inter alia*, that there was evidence of mild micronodular coal workers' pneumoconiosis which was insufficient to have altered pulmonary function or caused disability. EX 14 at 6-7. With respect to the etiology of Mr. Burns' emphysema, Dr. Oesterling wrote, in relevant part:

> [T]he medical records do afford a history of cigarette smoke inhalation specifically, a spirometric study performed at St. John's Hospital on 7-16-97 states in the history "The patient has been a 2 pack per day smoker for 20 years." This is a 40 pack year smoking history and more than adequate to explain his emphysematous change. Emphysema, once it is present, is irreversible, thus despite the suggestion that his smoking history was remote, it is still the etiology of this gentleman's emphysema.

Id. at 7. Dr. Oesterling also attributed fibrotic changes in both lungs primarily to hemosiderin, noting:

> [C]onfusion . . . has arisen in this case due to two pigments, hemosiderin and anthracotic pigment. The predominate fibrotic response throughout his lung sections is in response to hemosiderin and not mine dust. Thus it is unfortunate that the prosector has elected to extensively sample the lower lung field excluding the upper lobes. More careful sampling of the latter areas would have been far

> more beneficial in evaluating the presence or absence of coalworkers' pneumoconiosis and particularly in staging the level of that disease process which is present in the sections. . . .

Id. at 8.

In his March 23, 2005 supplemental report, Dr. Perper notes that, of the five pathologists who microscopically examined the autopsy lung sections, Dr. Oesterling was the only pathologist who diagnosed the presence of hemosiderosis. CX 8 at 11. He wrote:

> Not only Dr. Perper but the prosector Dr. O'Neil, and Dr[s]. Naeye and Caffrey did not observe or mention any increase in hemosiderin, histiocytes with hemosiderin or extensive fibrosis with hemosiderin qualifying for the diagnosis of hemosiderosis.

Ibid. Dr. Perper further noted that none of the other pathologists had, like Dr. Oesterling, diagnosed panlobular versus centrilobular emphysema. *Ibid*.

Dr. Oesterling's report was among the documents reviewed by Dr. Cohen as noted in his supplemental report of March 21, 2005. CX 7. Dr. Oesterling's opinion did not influence Dr. Cohen's opinion with respect to the cause of the Miner's pulmonary disability.

I find Dr. Oesterling's opinion is entitled to less weight than the contrary opinions of Drs. Perper and Cohen since his conclusion that the Miner's emphysema was caused by his smoking is clearly based on an erroneous assumption with respect to Mr. Burns' smoking history. As noted above, Dr. Oesterling wrote: "The patient has been a 2 pack per day smoker for 20 years." This is a *40 pack year smoking history* and more than adequate to explain his emphysematous change." EX 14 at 7 (emphasis added).

The evidence of record shows that Mr. Burns' smoking history was much less than 2 packs per day for 20 years. For example, the Miner's wife testified that Mr. Burns started smoking after he left home to enter the Navy and she and her husband both quit in 1953. Tr. 41. The Miner's daughter also testified that her father stopped smoking cigarettes around mid 1953. Tr. 34. When Dr. Thomson examined the Miner on November 5, 1986 on behalf of the Department of Labor, he noted a smoking history from 1941 to 1958 of 1 pack per day. DX 32. Dr. Sanjabi similarly performed an examination of the Miner on behalf of the Department of Labor on November 23, 1999. DX 6. He noted a smoking history of one pack per day beginning at age 21 and ending in the 1950's. The Miner was born November 8, 1922, DX 1, and thus would have started smoking, according to Dr. Sanjabi's report, sometime around 1943. Other medical records put the length of smoking at ten years or less. *See, e.g.,* smoking history taken by Dr. Tuteur reflecting 1 pack per day smoking between age 20 and 28 (DX 26); letter dated Jan. 31, 2001 from Dr. Moses to Dr. Travis stating "patient smoked for only about ten years, but he does have "black lung disease" and worked in the coal mines for many years exposed to considerable dust apparently." (DX 58; EX 7); Consultation report by Dr. Migone dated April 19, 2002 noting the Miner "quit smoking in 1950." (DX 58; EX 5).

For purposes of this decision, I give the greatest weight to the testimony of the Miner's wife and daughter, especially in light of the fact that Mrs. Burns recalled that she and her husband quit smoking together in 1953. Thus, even if Mr. Burns started smoking 1 pack of cigarettes per day *immediately* after entering the Navy in December 1941, and stopped smoking in December 1953, he would have only a 12 (versus 40) pack year smoking history. I therefore accord Dr. Oesterling's opinion less weight. *See, e.g., Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993) (physician's opinion less probative where based on inaccurate smoking history).

Opinion of Dr. Repsher

After reviewing medical evidence submitted to him by Employer's counsel, Dr. Repsher authored a report dated April 12, 2004 in which he concluded, *inter alia*, that the Miner had pathological evidence of simple coal workers' pneumoconiosis as well as mild to moderately severe mixed COPD. EX 17 at 14. He further concluded that "Mr. Thomas Burns had simple histologic coal workers' pneumoconiosis, but he did not suffer from during his life nor did he die, as a result of any pulmonary respiratory condition, either caused by or aggravated by his employment in the coal mining industry." *Id.* at 15. Reasons given for this opinion included, *inter alia*, that the Miner's mild to moderately severe obstructive ventilatory impairment was not due to coal dust exposure. *Ibid*. He wrote in relevant part:

> The inhalation of coal mine dust may cause COPD, but the average impairment is so small as to not be discernible in an individual miner, since it is much less than the anticipated test-to-test and day-to-day variation in results. It is discernible only in a statistical sense, when comparing a large group of dust-exposed coal miners with another large group of non dust-exposed workers in other industries.

Ibid.

Dr. Perper, in his March 23, 2005 supplemental report, and Dr. Cohen, in his March 21, 2005 supplemental report, criticized various aspects of Dr. Repsher's opinion. CX 7, 8. Dr. Cohen noted. in particular. that Dr. Repsher, unlike Dr. Cohen, failed to evaluate the actual measured values for all Mr. Burns' pulmonary function studies with the standard set of predicted values recommended by the American Thoracic Society and the American Medical Association to ensure uniformity of the interpretations. CX 7 at 2. For several reasons, Dr. Cohen further disagreed with Dr. Repsher's conclusion that a comparison between pulmonary function test results and other data "allows one to totally disregard the risk factors associated with 40 years of coal dust exposure and then speculate that this patient's pulmonary impairment might have been related to heart disease, coronary artery disease or other acute medical events." *Ibid*. He noted, for example, that medical records substantiate that Mr. Burns was diagnosed with and treated for both lung and heart disease and that a 1999 bronchoscopy resulted in a diagnosis of bronchiectasis and black lung disease. *Ibid*. He further stated that his cardiologist believed in 2001 that his primary problem was lung disease, and his treating pulmonary specialist noted that Mr. Burns' lung condition worsened. *Ibid*. Finally, he noted that the Miner's pulmonary impairment was primarily obstructive and heart disease does not cause obstruction. *Ibid*.

I find the opinions of Drs. Cohen and Perper better documented and reasoned than the contrary opinion of Dr. Repsher. In particular, I note that Dr. Repsher's conclusion that the "average impairment [from COPD caused by the inhalation of coal mine dust] is so small as to not be discernible in an individual miner. . . ." is, like the opinions of Employer's other experts, clearly inconsistent with the prevailing view in the medical community as described in the studies cited in the Department's Final Rule. In rejecting similar opinions, the Department noted, *inter alia*, that it had

> reviewed all of the medical and scientific evidence referenced in the rulemaking record, and does not agree that the record lacks valid support for the proposition that coal mine dust exposure can cause obstructive pulmonary disease. The Department's position is fully supported by NIOSH, the statutory advisor to the black lung benefits program, which responded favorably to the Department's proposed revisions. The considerable body of literature documenting coal mine dust exposure's causal effect on the development of chronic bronchitis, emphysema and associated airways obstruction constitutes a clear and substantial basis for this aspect of the revised definition of pneumoconiosis.
>
> The term "chronic obstructive pulmonary disease" (COPD) includes three disease processes characterized by airway dysfunction: chronic bronchitis, emphysema and asthma. Airflow limitation and shortness of breath are features of COPD, and lung function testing is used to establish its presence. Clinical studies, pathological findings, and scientific evidence regarding the cellular mechanisms of lung injury link, in a substantial way, coal mine dust exposure to pulmonary impairment and chronic obstructive lung disease. In discharging its congressionally-mandated duty to recommend a permissible exposure limit for coal mine dust, NIOSH conducted a comprehensive review of the available medical and scientific evidence addressing the impact of coal mine dust exposure on coal miners. . . . NIOSH concluded that "[i]n addition to the risk of simple CWP and PMF [progressive massive fibrosis], epidemiological studies have shown that coal miners have an increased risk of developing COPD."

Final Rule, 65 Fed. Reg. at 79939. I therefore find that Dr. Repsher's opinion is entitled to diminished weight.

Opinion of Dr. Fino

In his May 6, 2004 consultation report, Dr. Fino stated that he reviewed extensive medical records of Mr. Burns submitted to him by Employer's counsel. EX 18. In his discussion of the medical evidence, Dr. Fino noted, *inter alia*, that: there was sufficient evidence of coal workers' pneumoconiosis pathologically; the miner stopped working in the mines in 1985 after 40 years; and "[o]ne year after he left the mines, in 1986, he had absolutely normal spirometry." *Id.* at 13. Dr. Fino further noted that the next series of pulmonary function studies performed eleven years later showed obstructive ventilatory abnormality, but said "[i]t would be unusual to develop new obstructive lung disease when you leave the mines, if one were going to

attribute the obstruction to coal mine dust." *Ibid*. He opined, *inter alia*, that coal workers' pneumoconiosis did not contribute to the Miner's disability. *Id.* at 17.

With respect to the issue of whether the Miner was totally disabled by a respiratory or pulmonary impairment related to coal mine employment, I find Dr. Fino's opinion of little value. Although, as noted above, he stated that coal workers' pneumoconiosis did not contribute to Mr. Burns' disability, he gave no rationale, nor did he cite to any particular objective test results, which support that conclusion. I therefore give the opinion little weight.

Based on all the foregoing, I find that the weight of the medical evidence supports the conclusion that the Miner's pulmonary impairment was, prior to his death, totally disabling and that his pneumoconiosis was a contributing cause with respect to his disability. I thus find that the Claimant, on behalf of the deceased Miner, is entitled to benefits.

Survivor's Claim of Mrs. Burns

Mrs. Burns' claim, unlike that of her deceased husband, is subject to the Department's amended regulations which took effect on January 19, 2001, and any decision regarding this claim must therefore be based on evidence which comports with the evidentiary limitations set forth in 20 C.F.R. § 725.414.[16] With respect to Claimant's entitlement to benefits, Section 718.205 of the regulations provides that benefits are available to eligible survivors[17] of a miner whose death was due to pneumoconiosis. An eligible survivor will be entitled to benefits if any of the following criteria are met:

1. Where competent medical evidence establishes that the miner's death was due to pneumoconiosis;

2. Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death, or where death was caused by complications of pneumoconiosis; or

3. Where the presumption set forth in § 718.304 (evidence of complicated pneumoconiosis) is applicable.

20 C.F.R. § 718.205(c).

Pneumoconiosis is a substantially contributing cause of a miner's death if it hastens the miner's death. 20 C.F.R. § 718.205(c)(5). The United States Court of Appeals for the Seventh Circuit, within whose jurisdiction this case arises, has held that a miner's death is considered to

[16] As the Board noted in *Church v. Kentland-Elkhorn Coal Corp.*, BRB Nos. 04-0617 BLA and 04-0617 BLA-A (Apr. 8, 2005) (unpub.), medical evidence submitted in a miner's claim is not automatically admissible in a survivor's claim filed after January 19, 2001. The Board further concluded that the medical evidence from the living miner's claim must meet the limitations under 20 C.F.R. § 725.414 to be considered in the survivor's claim and medical opinion evidence in the survivor's claim should consider only evidence that is properly admitted. *Ibid*.

[17] Claimant was married to the Miner on February 1, 1946 and remained married to him until his death on February 16, 1999. DX 1, DX 38. Employer does not contest, and I find, that she is an eligible survivor.

be due to pneumoconiosis where pneumoconiosis hastens, even briefly, the miner's death. *See Peabody Coal Co. v. Director, OWCP* [Railey], 972 F.2d 178, 16 BLR 2-121 (7th Cir. 1992).

A threshold determination in a survivor's claim is the existence of pneumoconiosis. *See* 20 C.F.R. §718.205(a)(1)*; Trumbo v. Reading Anthracite Co.*, 17 BLR 1-84 (1993). The existence of pneumoconiosis caused by the Miner's coal mine employment is not, as noted above, contested by Employer and is clearly established by the evidence of record. *See* Emp. Br. at 54. Evidence with respect to the issue of causation of Mr. Burns' death is set forth below.

According to the Miner's death certificate, signed by Dr. Ana O. Migone, Mr. Burns died on June 7, 2002 in Carbondale, Illinois at the Memorial Hospital of Carbondale. DX 38. The immediate cause of death was listed as right lower lobe pneumonia, with pneumoconiosis noted as a condition which gave rise to the immediate cause of death. Other significant conditions contributing to death but not resulting in the underlying cause of death were noted as diabetes, atrial fibrillation, and heart disease. *Ibid*.

An autopsy of the Miner was performed on June 7, 2002 by Dr. Patrick O'Neill, limited by the Miner's wife to the lungs. DX 41. With respect to his internal exam, Dr. O'Neill noted, *inter alia*, extensive fibrinous adhesions of the pulmonary pleural surfaces and marked black anthracotic pigment deposition. *Ibid*. With respect to his microscopic examination, Dr. O'Neill noted that the left lower lobe of the lung showed fibrosed granulomas with central necrosis and large numbers of histiocytes containing black anthracotic pigment within the fibrous tissues. There was also dilation of alveolar spaces in the right and left lung tissues representing emphysematous change and scattered fibrous nodules containing intense anthracotic pigment. Dr. O'Neill noted slender white crystals on polarization consistent with silica and stated that the consolidated areas of the right lower lobe showed fibrin, neutrophils and lymphocytes filling, expanding and replacing the alveolar spaces representing a pneumonic process. His final diagnoses included coal workers' pneumoconiosis, pneumonic consolidation in the right lower lobe, marked pleural adhesions bilaterally, findings consistent with clinical history of severe COPD, and a 17.0 cm midline chest scar consistent with clinical history of aortic valve replacement.

The x-ray, CT scan, and other medical evidence relevant to this survivor's claim has been thoroughly discussed above. Likewise, the medical opinions of Drs. Perper, Cohen, Tuteur[18] and Naeye have been discussed in the context of whether Mr. Burns was, while alive, totally disabled by a pulmonary condition caused by his coal mine employment. The conclusions of these four physicians regarding the cause of the Miner's death are further described below.

[18] Dr. Tuteur submitted two medical reports, one dated March 7, 2005 (EX 21A) discussed above and one dated March 9, 2005 (EX C). The substantive content and opinions contained therein are substantially the same, although the evidence considered by Dr. Tuteur with respect to the Widow's claim (EX C) is more limited since this claim is subject to the evidentiary limitations set forth at 20 C.F.R. § 725.414. References in this portion of the Decision and Order will be to EX C.

Opinion of Dr. Perper

Dr. Perper opined, based on his review of the medical reports, treatment records, and tissue slides, that "coal workers' pneumoconiosis was a significant and primary contributory cause of death of Mr. Burns (along with his other contributing cause of death such as his arteriosclerotic cardio-vascular) and a hastening factor of his death, both directly and indirectly . . . ." DX 68 at 35. He based this conclusion on the following findings:

1. Normal lung tissue had been directly replaced by pneumoconiotic lesions and associated centrilobular chronic emphysema and resulting hypoxemia

2. The Miner suffered from complicating acute bronchopneumonia, acute exacerbations of pulmonary conditions are common, and silica found in the pneumoconiotic lesions of Mr. Burns increased his susceptibility to pulmonary infections.

3. Mr. Burns' hypoxemia is known to be a precipitating and aggravating condition with respect to cardiac arrhythmia in individuals, such as the Miner, with heart disease.

Ibid.

Opinion of Dr. Cohen

According to Dr. Cohen, "[a]lthough [Mr. Burns] had heart disease, the medical records and autopsy results would support the conclusion that it was not as prominent or untreatable as the lung disease." CX 5 at 30. Dr. Cohen further wrote:

> Mr. Burns had severe simple pneumoconiosis as well as severe obstructive lung disease due to his coal mine dust exposure. He had severe obstruction and diffusion impairment and gas exchange impairment near the time of death, an indication of substantial and clinically significant interstitial and obstructive lung disease. I have no doubt that his coal mine dust exposure was a primary cause or substantial contribution to the development of his severe impairment, his medical pneumoconiosis and his legal pneumoconiosis, all of which significantly contributed to his respiratory death.

Ibid.

Opinion of Dr. Tuteur

Dr. Tuteur opined that, "[n]either coal workers' pneumoconiosis, nor any other coal mine dust-induced disease process contributed to, caused, or hastened Mr. Burns' death from advanced ischemic cardiomyopathy and the multiplicity of complications of the disease and its treatment." EX C at 8. Dr. Tuteur acknowledged that the Miner had pulmonary abnormalities, but concluded that these pulmonary abnormalities were unrelated to the inhalation of coal mine

dust or the development of coal workers' pneumoconiosis. *Id.* at 9. He attributed Mr. Burns' pulmonary abnormalities instead to thoracic trauma from the Miner's 1989 automobile accident and complications associated with his coronary artery bypass graft and aortic valve replacement. *Ibid*.

Opinion of Dr. Naeye

Dr. Naeye concluded that there were minimum findings sufficient to make the diagnosis of simple coal workers' pneumoconiosis in the lungs of Mr. Burns, but that "[t]he size and extent of these lesions are far too small for them to have had any measurable effect on lung function, so they could not have caused any disability or contributed in any way to this man's later death." EX D at 2. He further determined that "[c]igarette smoking had the dominating role in causing his chronic bronchitis, progressive centrilobular emphysema and death." *Id.* at 3.

For many of the reasons discussed with respect to the Miner's claim, I find the opinions of Drs. Tuteur and Naeye less persuasive than the contrary opinions of Drs. Perper and Cohen. For example, while Dr. Tuteur attributes Mr. Burns' pulmonary impairment to the trauma from the Miner's 1989 automobile accident and cardiac problems following surgery, the opinions of Mr. Burns' treating pulmonologist and cardiologist, as Dr. Cohen noted, are contrary to that opinion. Likewise, Dr. Tuteur failed to explain his conclusions that Mr. Burns did not have any restrictive pulmonary impairment and his *obstructive* pulmonary impairment was due in part to the 1989 accident in light of the fact that, as Dr. Cohen further noted, any impairment in pulmonary functioning caused by such accident would manifest itself as a *restrictive*, rather than obstructive, impairment. Similarly, Dr. Tuteur, like Dr. Naeye, gives little credence to the proposition that prolonged exposure to coal dust may cause significant obstructive lung disease. As noted above, the prevailing view in the medical community, cited and relied on by the Department of Labor in adopting the revised regulations, is to the contrary. Furthermore, neither physician adequately explained how Mr. Burns' limited and remote smoking history, which ended in 1953, could be considered a significant factor in the development of the Miner's pulmonary impairment which first became manifest several years after he stopped coal mining in 1985.

In contrast to the opinions of Drs. Tuteur and Naeye, Drs. Perper and Cohen both attributed Mr. Burns' debilitating pulmonary impairment principally to his 40-year history of exposure to coal dust while working in the coal mining industry. Both physicians recognized that such exposure can, and in the Miner's case did, cause both clinical and legal pneumoconiosis. Both physicians further concluded that Mr. Burns' compromised pulmonary condition was a substantial contributing cause of the Miner's death on June 7, 2002. These conclusions are consistent with the opinion of Dr. Patrick O'Neill, the autopsy prosector, as well as that of Dr. Ana Migone, the physician who signed Mr. Burns' death certificate.[19] They are

[19] Dr. Migone not only signed the Miner's death certificate but was the attending physician during Mr. Burns hospitalization at various times at Memorial Hospital. *See, e.g.*: Discharge summary dated February 26, 2002 noting that attending physician was Ana Migone (DX 58/(EX 5 at 457); hospitalization records dated March 28, 2002 where Dr. Migone diagnosed, *inter alia*, black lung disease and mitral insufficiency (*Id*. at 225); hospitalization records dated April 18, 2002 where Dr. Migone and Dr. Salmon diagnosed, *inter alia*, history of chronic obstructive pulmonary disease contributing to congestive heart failure exacerbation, and pneumonia (*Id*. at 106); admitting

further supported by the opinions and conclusions of Dr. Crabtree, the Miner's treating pulmonary specialist, and Dr. Moses, his treating cardiologist.

Based on all the foregoing, I find that the weight of the medical evidence supports the conclusion that Mr. Burns' pneumoconiosis was a substantially contributing cause of his death. I thus find that Claimant is entitled to an award of benefits as the Miner's eligible survivor.

Attorney's Fees

No award of attorney's fees for services to Claimant is made herein, as no application has yet been received from her representative. Thirty days are hereby allowed to Claimant's counsel for the submission of such application. Her attention is directed to §§ 725.365 and 725.366 of the Regulations. A service sheet showing service upon all parties, including the Claimant, must accompany the application. Parties have fifteen days following receipt of such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

The claims of MILDRED E. BURNS, survivor of and on behalf of THOMAS BURNS, for benefits under the Act, are hereby GRANTED, and it is hereby ORDERED that CONSOLIDATION COAL COMPANY, the Responsible Operator, shall pay to Claimant all benefits to which she is entitled under the Act, commencing October 22, 1999 with respect to the Miner's claim and July 15, 2002 with respect to the Survivor's claim.

A

STEPHEN L. PURCELL
Administrative Law Judge

Washington, D.C.

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. §725.481, any party dissatisfied with this Order may appeal it to the Benefits Review within 30 days from the date of this Order by filing a Notice of Appeal with the Benefits Review Board, P.O. Box 37601, Washington DC 20013- 7601. A copy of a Notice of Appeal must also be served on Donald S. Shire, Esq., Associate Solicitor for Black Lung Benefits. His address is Room N-2117, Frances Perkins Building, 200 Constitution Avenue, N.W., Washington DC 20210.

---

summary prepared by Drs. Somers and Migone dated June 5, 2002 noting history of black lung and increase in symptoms with some hemoptysis (expectoration of blood). *Id.* at 8.